Gynaecology Essay Questions

Lymph Notes

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Gynecology:

GENERAL SCHEMES

ليه الschemes دي مهمة؟ عشان اللي أهم من الشغل .. تظبيط الشغل #مندوهيات ☺

1. ***"A quick introduction to any question is always a good idea"***

Examples:

Abnormal uterine bleeding is a condition which describes any bleeding apart from the normal pattern (which is usually cyclic occurring every 3-5 weeks and duration 3-5 days). It is one of the commonest complaints in gynaecological practice.	**Fibroids** are the commonest benign tumors of the female genital tract (up to 20% of women in CBP) They are most commonly corporeal (95%) and much less commonly cervical or broad-ligamentary.

2. How to answer "Discuss" ..

1. ***Definition.***
2. ***Incidence:***

Common or rare

3. ***Classification.***

±

4. ***Etiology:***
   - Definite cause
   - No definite cause → theories & predisposing or risk factors.
5. ***Pathogenesis:***

   ± (How the etiology causes the disease?).
6. ***Pathology:***
   - Gross picture.
   - Microscopic picture.
   - Grading.
   - Spread.
   - Staging.
   - Complications.

N.B.

- "If benign tumor: write all except grading, spread, staging"

7. ***Diagnosis: (C/P + Investigations)***
   - Type of the patient.
   - Symptoms (history).
   - Signs (general, abdominal, local).
   - Investigations.
   - Differential diagnosis يؤلف

N.B.

- Add staging in case of malignancy.
- C/P: All except investigations and staging.

8. ***Treatment:***
   - Prophylactic (# predisposing factors).
   - Active.
   - Prognosis (complications).

N.B.

- Add ttt of complications in active ttt.
- Management = Diagnosis & treatment.

3. How to answer "evaluate" or "discuss management of" questions, with vague titles ..

For example:

- Evaluate a case of 2ry amenorrhea
- Evaluate a case of a case of AUB
- Evaluate a case of vaginal discharge

There's a general scheme:

1. History (Each part of general sheet is important)
2. Examination (General - Abdominal - Local)
3. Investigations (and they are always US, laboratory, Radiological, invasive (endoscopy and biopsy)

Example:

2ry Amenorrhea	
History	**Personal:** Age (POF?) Gravity and parity (la7san teb2a lessa walda w 3andaha sheehan aw 7aga) Special habits (athlete?)
	Present: Duration? Precipitating event (stress? Menstrual irregularities?) Other symptoms (pelviabdominal swelling w positive pregnancy symptoms? Galactorrhea? Hirsutism?)
	Obstetric: Abortion? Puerpural sepsis? (Asherman)
	Contraceptive: IUCD and inf? COCs (post pill amenorrhea)
	Menstrual: LMP, irregularities? Anovulation?
	Past: Other drugs? Thyroid? DM? TB?
	Family: DM and hyperlipidemia (PCOS)
Examination	**General:** Obesity, acanthosis negricans (signs of insulin resistance? PCOS Hirsutism? Degree (mild PCOS severe tumor!) Galactorrhea? Goitre? Weight and body fat? (Too low?)
	Abdominal: Pelviabdominal swelling (pregnant!) momken ovarian tumor?
	Local: Pregnancy changes? Atrophic changes? (Premature Menopause) virilization (androgen secretng tumor?) Bimanual, ovarian mass?

Investigations (If you cannot remember specific investigation scheme., the following is also correct ..)	**US:** Pregnancy? Ovarian tumor? Atrophic endometrium (menopause)
	Laboratory: hCG (ahamm 7aga) FSH, LH, PRL, TSH, E2, Androgen levels W momken blood sugar and lipid levels (PCOS)
	Radiological: (law shakek f pituitary or hypothalamic causes)
	Invasive: Hysteroscopy for Ashermann Laparoscopy for PCOS

N.B.

Scheme for last part of book (imaging - endoscopy - operations - instruments):

See page 146

Essay Questions:

1. ANATOMY

1. Bartholin gland (function –anatomy –complications) (sep 2009-2014)

Anatomy:

- Bartholin glands are bilateral compound racemose glands situated deep in the labia majora, at the junction of its posterior and middle third.
- Its duct is 2 cm long and opens between the hymen and labium minus

Function:

They secrete mucus during sexual excitement to act as a lubricant

Complications:

1. If the duct is obstructed it may from a vulva swelling (bartholin cyst – abscess)
2. The gland rarely is a site for a Bartholin's gland tumour

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2. Anatomical relations, support, blood supply and lymphatic drainage of vagina (june 2016)

Anatomical relations:

A. *Anteriorly*:
Trigone of urinary bladder in upper 1/3 and urethra in the lower 2/3

B. Posteriorly:
Peritonem of pouch of doughs in the upper 1/3, the ampulla of the rectum in the middle 1/3 and the perineal body in the lower 1/3

C. *Laterally:*
- lower end : Bulbo cavernosus muscle, vestibular bulb and Bartholin's gland
- 1 cm above orifice: urogenital diaphragm
- 2.5 cm above the orifice: levator ani muscle with pelvic fascia above it
- The lateral fornix gives attachment to the lower part of the cardinal ligaments
- The ureters pass through cardinal ligaments 1 cm lateral to vagina

[A side note: Notice that the pouch of douglas lies at a lower level (upper 1/3 of the vagina) than that of the uterovesical pouch (related to the body of the uterus), this may be due to the fact that the bladder is more distensable (evacuted 2-4 times/day) than the rectum (evacuated less frequent) making the covering peritoneum hanging at a higher level,
The anatomy serves the function & the function shapes the anatomy
Also,the douglas pouch is the most dependable part of the peritoneal cavity, so it will be the site of accumulation of any fluid or pus]

Vaginal supports:

1. Ligaments attached to the upper vagina
- Pubocervical ligament anteriorly
- Mackenrodt's ligament laterally
- Uterosacral ligament posteriorly

2. Levator ani muscle:
Pubovaginalis part of pubococcygeus

3. *Triangular ligament and perineal membrane.*
4. *Vaginal fascia:*

   CT fascia that condenses anteriorly forming the vesicovaginal fascia and posteriorly forming recto-vaginal fascia

Blood supply

A. *Arterial supply:*
   - The vaginal artery "from internal iliac" is the main blood supply to the supper vagina
   - Additional branches to lower vagina from:
     - Middle rectal artery (from internal iliac artery)
     - Inferior rectal artery (from internal pudendal of internal iliac)

B. *Venous drainage:*

   Veins from the vaginal plexus connect with the plexus around the bladder and the rectum and drain into the internal iliac vein by veins that accompany their corresponding arteries.

Lymphatic drainage:

- Upper 1/3 follows lymphatic drainage of the cervix which is:
  - Primary groups:Paracervical,parametrial,obturator,internal & external iliac LNs
  - Secondary groups:Common iliac, lateral sacral & para aortic LNs
- Lower 1/3 drains to the inguinal LN
- The middle 1/3 drains in both upper and lower direction

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3. Lymphatic drainage of uterus (Sep. 2011, 2009, June 2012)

1. *Fundus:*

   To the para- aortic lymph needs via ovarian vessels

2. *Cornu:*

   To the superficial inguinal lymph nodes via lymphatic's of the round ligament.

3. *Body:*

   To the internal then external iliac lymph nodes via uterine vessels.

4. *Isthmus:*

   As that of the cervix.

5. *Cervix:*

   Two groups of lymphatics:

   a. Primary groups: Para cervical, parametrial, obturator, internal and external iliac nodes.
   b. Secondary groups: common iliac Para-aortic and lateral sacral lymph nodes.

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4. Blood supply & lymphatic drainage of the uterus (june 2015/sep 2013, 2016)

Arterial Supply:

- The uterine arteries from the main supply of uterus, they arise from anterior division of internal iliac artery and run medially towards the isthmus , in the base of broad ligament cross above ureter 1/2 an inch lateral to supra vaginal cervix.
- At the level of internal as each artery gives an ascending and descending branch, with their contralateral fellows they from the circular arteries which supply the uterus and the supper cervix:

a. <u>The ascending branches:</u>

Pass upward in tortious manner parallel to the lateral of the uterus between the 2 layers of broad ligament to end by anastomosing with branches of the ovarian arteries near the uterine cornu

b. <u>The descending branches:</u> (supply the lower cervix)

- The main branch, along its length divides into anterior and posterior coronary arteries that surround the uterus circumferentially thus uterus is least vascular in the midline.
- The coronary artries give rise to the radial arteries, that penetrate the myometrium to end as the basal arteries supplying the endometrium.

Venous drainage:

Starts as a plexus between the 2 layers of the broad ligament "pampiniform plexus" that communicate with vesical plexus and drains into uterine and ovarian veins.

Lymphatic drainage:

As previous question.

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5. Anatomy of uterine ligaments (1999)

❖ Ligaments attached to the uterine fundus and body an each side include: the broad ligament, the round ligament and the ovarian ligament.

❖ These ligaments being soft, lax and easily stretchable, play a very limited or almost no role in pelvic support of the uterus in contrast to the strong, tough cervical ligaments

1. *The broad ligament:*

- It is a double sheet of peritoneum that extends from the lateral wall of uterus to the lateral pelvic wall.
- Its outer upper part forms the infundibulo pelvic ligament in which ovarian vessels traverse their way to the ovay.

Contents of broad ligament:

a. The Fallopian tubes.

b. The round ligament, the ovarian vessels, the uterine vessels, the ureter, parametrial lymphatics and lymph nodes, sympathetic and parasympathetic nerves, parametrial pelvic cellular tissue and fascia.

(A side note: They are arranged from above downwards)

c. <u>Embryological remnants of the Wolffian ducts:</u>

- Hydatid cyst of morgagni:

  A pea sized thin walled cyst attached to the fimbria and of the tube.
- Koblet's tubules:

  Blind tubules lying on outer aspect of the ligament.
- Epoophron:

  Tubules lying between the ovary and the TUBE.
- Paroophoron:

  Tubules lying between the ovary and the uterus.
- Gartner`s duct:

  Runs in the broad ligament parallel to the tube then downwards lateral to uterus then anterolateral to vagina.

"Applied anatomy":

Remnants of wolffian duct may undergo cystic dilatation giving rise to:-

- A Parovarian cyst that lies between leaf of the broad ligament.
- Gartner`s duct cyst in the antero lateral wall of the vagina.

2. *The round ligament:*
    - This is fibro muscular ligament attached to the uterine cornu.
    - It runs downwards and forwards in between the 2 leafs of the broad ligament to enter the inguinal canal via the internal inguinal ring and comes out of it at the external inguinal ring to be inserted in upper part of the labia majora.
    - Function:

      It pulls the uterus forwards and help keeping it in an anteverted position

"Applied anatomy":

Lymphatics accompanying the round ligament drain the cornu of uterus to superficial inguinal LN.

3. *The ovarian ligament:*
    - It is fibromuscular ligament that attaches to inner lower pole of the ovary to the cornu of the uterus.
    - It plays no role in pelvic support of the uterus.

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6. Anatomy of the cervical ligaments (June 2014)

- The cervical ligaments are condensed thickening of the pelvic cellular tissue, that lies between the pelvic peritoneum above and the levantor ani below and radiates outwards from the cervix to reach the pelvic walls.
  these strong ligament , that contain few smooth muscle fibers, act as the chief support of the uterus and pelvic structures.
- 3 pairs of ligaments can be distinguished, which extend from the supravaginal part of the cervix and upper part of the vagina to the pelvic walls:
    1. *Mackenrodt`s ligaments "the cardinal ligament of the cervix":*

       Spread out on either side from the lateral surface of the cervix and vagina, in a fan shapped manner, and are inserted in the lateral pelvic wall.
    2. *Utero sacral ligament:*

       From the posterior aspect the cervix and vagina, backwards surrounding the rectum, below the utero – sacral folds of peritoneum , to become inserted in the third piece of the sacrum
    3. *Pubo – cervical ligaments:*

       Extend from the anterior surface of the cervix and vagina, forwards beneath the bladder and surrounding the urethra, the posterior surface of the pubis.

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7. The ureter is at risk point during gynecological surgery. Give two common sites for injury (june 2009)

1. On clamping the infundibulo pelvic ligament where the ureter pass below ovarian vessels
2. On clamping the uterine artery as it passes below it 1 cm lateral to cervix
3. During clamping the vaginal angles and the parametrium 1 cm lateral to vaginal vault

Also:

- Avascular necrosis occurs due to cutting of blood vessels in Wertheim's operation

- Ischemic necrosis due to over dose of radiation in ttt of cancer cervix

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8. Pelvic part of ureter (sep 2012)

The ureter is a narrow muscular tube, 25 cm long, runs retro-peritoneally from kidney to bladder. Its wall undergoes peristaltic movement to propel urine downwards.

- *At pelvic inlet:*
  Enters pelvis **above** the bifurcation of common iliac artery **anterior** to sacroiliac joint
- *In pelvis:*
  - Runs Downwards in front of internal iliac artery
  - At base of broad ligament runs medially and forwards through parametrium till 1cm lateral to supra vaginal cervix then below and at a right angle to uterine artery
  - The ureter then passes forward through the ureteric canal in the upper part of cardinal ligament, closely related to lateral vaginal fornix to enter trigone of urinary bladder.

Blood supply of the ureter:

Supplied by branches from the internal iliac artery, the uterine artery, the inferior vesical artery and vaginal artery.

Applied anatomy:

See before (sites of ureteric injury during hysterectomy).

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9. Enumerate ovarian attachments (sep 2008/aug 2009)

Fixed in the pelvis by 3 attachments:

1. Mesovarium: a peritoneal fold that sispends the ovary to the back of broad ligament.
2. The infundibulo-pelvic ligament: suspends upper pole of the ovary to the lateral pelvic wall & carries ovarian vessels, nerves, lymphatics.
3. The ovarian ligament: attaches lower pole to the cornu of uterus.

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2. ENDOCRINOLOGY

Puberty

1. Define puberty and its clinical manifestations.

Definition:

- The period of life that marks the normal physiologic transition from childhood to sexual and reproductive maturation (physical, mental & sexual growth).
- Hypothalamus, pituitary and ovaries undergo a maturation process leading to:
  - Development of secondary sexual characters (breast, sexual hair, and genitalia).
  - Limited acceleration in physical growth.

Age:

- Start by the age of 8-9 years.
- Complete by the age of 12-14 years (onset of menses and acquiring reproductive capacity).

Clinical manifestations:

1. *Growth spurt:*
   - The **1st sign** of puberty.
   - Peak growth velocity at the age of 11 years.
   - Followed by slower growth rate until cessation (by the age of 15 years); due to closure of bone epiphysis under effect of increased estrogen.
2. *Thelarche (breast development):*

   5 phases under effect of **estrogen**, including:
   - Growth of the body of breast (budding).
   - Pronounced areolar development.
   - Full breast development (breast tissue grows to be confluent with the areola).
3. *Pubarche:*
   - Growth of pubic hair under effect of **ACTH** and **androgens**.
   - 5 stages according to degree of distribution.
4. *Menarche:*
   - Onset of 1st menstruation.
   - Average age in Egypt: 12.5 years.
5. *Axillarche:*

- Growth of axillary hair.
- Appears later under effect of **androgens**.

Tanner stages of pubertal growth:

Pubertal cascade:

1. Starts by growth spurt.
2. Breast development.
3. Pubic hair.
4. Menarche.
5. Finally, axillary hair.

Tanner classified physical stages of puberty into 5 stages depending on degree of breast

development and distribution of pubic hair.

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2. Etiology and treatment of delayed puberty.

Definition:

- No secondary sexual characters are noted by the age of 13-14 years.

Or

- If menses is still absent by the age of 15-16 years.

Incidence:

3%.

Causes, diagnosis and management (see primary amenorrhea for details).

Etiology:

"Same as primary amenorrhea + constitutional delay of puberty + excessive weight loss + pituitary damage".

1. *Hereditary factors:*
   - 10-20% of cases.
   - Constitutional delay may run in families due to delayed activation of GnRH pulse generator.
2. *Chromosomal abnormalities, genetic and autoimmune disorders:*
   - 50% of cases.
   - Ovaries are non functioning and unable to respond to gonadotropins, leading to marked elevation in FSH & LH (**hypergonadotropic hypogonadism**).
   - Examples:
     - Turner syndrome (45, XO).
     - Premature ovarian failure (POF).
     - Autoimmune ovarian failure.
     - Gonadal dysgenesis with a Y chromosome present (testicular-feminization syndrome).
3. *Pituitary and hypothalamic tumors and severe illness:*
   - 10-15 % of cases.
   - Ovary is normal but there is lack of production of GnRH by hypothalamus leading to low FSH & LH levels (**hypogonadotropic hypogonadism**).
   - Examples:
     - Kallmann syndrome (congenital GnRH deficiency + anosmia).
     - Hypothalamic suppression by stress, severe diseases or malnutrition.
4. *Pituitary tumors and lesions:*
   - Prolactinoma and empty-sella syndrome induce hyperprolactinemia that leads to suppression of GnRH pulses.
   - Tumor invasion by craniopharyngioma suppress pituitary gonadotropins (**hypogonadotropic hypogonadism**).
5. *Congenital Mullerian agenesis or dysgenesis:*

   Absent or hypoplastic uterus with normal ovaries leading to primary amenorrhea with normal 2ry sexual characters (**eu-gonadotropic hypogonadism**).

Diagnosis and investigations:

Discuss as in examination and investigations of precocious puberty and primary amenorrhea.

Treatment:

According to the cause:

- If hereditary, usually no treatment is necessary.
- In some cases, hormone therapy (combined estrogen and progesterone) to stimulate development of secondary sexual characters.
  Or surgery to correct anatomical problems.
- In testicular feminization syndrome, gonadectomy must be done to prevent neoplastic transformation.

+ Discuss treatment of causes of primary amenorrhea.

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3. Precocious puberty.

Definition:

- Appearance of sexual characters before 8 years of age, with or without onset of menstruation.
- Leads to:
  - Psychological impact.
  - Early estrogen production carries the risk of short stature (early epiphyseal closure).

Types:

1. *Isosexual precocious puberty (IPCP):*

   Secondary sexual characters in agreement with genetic and phenotypic sex.

   A. *True IPCP (GnRH-dependent, possibly associated with ovulation):*

      i. *Constitutional:*
      - 74% of cases.
      - Idiopathic premature activation of the gene in GnRH nucleus, leading to early onset of FSH and estrogen secretion.

      ii. *Organic:*
      - CNS lesions that impede neural signals that normally inhibit GnRH release in childhood.
      - Examples:
        - Tumors: craniopharyngioma, adenoma, glioma.
        - Infections: meningitis, encephalitis.
        - Malformations: hydrocephalus, empty sella syndrome, hamartoma.
        - Head trauma.

   B. *Pseudo IPCP (GnRH-independent, not associated with ovulation):*

      i. Estrogen-secreting ovarian tumors (granulosa-theca cell tumors).

      ii. Primary hypothyroidism: elevated TSH may induce FSH production.

      iii. Exogenous estrogen intake.

2. *Heterosexual precocious puberty (HPCP):*

   Secondary sexual characters are in disagreement with genetic and phenotypic sex.

   A. Androgen-secreting tumors (virilizing ovarian or adrenal neoplasia).

   B. Congenital adrenal hyperplasia (CAH).

   C. Exogenous androgen intake.

3. *Isolated premature pubertal events:*
   - As premature menarche only, thelarche only, … etc. without other estrogen-induced

pubertal events or advancement of bone age.

- Usually benign with no need of treatment.

Diagnosis of precocious puberty:

1. *History and physical examination:*

   Age, height, growth spurt, breast development, and hair distribution, according to Tanner stages of pubertal development.

2. *Hormonal assay:*
   - Pituitary gonadotropins (serum FSH & LH).
   - Serum E_2, and DHEA to detect ovarian and adrenal causes.
   - Thyroid function tests (TSH, T_3 & T_4) to exclude primary hypothyroidism.
3. *Radiological investigations:*
   - X-ray wrist (non-dominant hand) to determine bone age.
   - CT & MRI for brain tumors and intracranial masses.
   - Pelvic US for exclusion of ovarian tumors or cysts.

Management:

Aim:

- Slowing down accelerated growth.
- Inducing regression of secondary sexual characters by reducing pituitary and ovarian hormones to avoid premature epiphyseal closure, which predisposes to short stature.

1. *Idiopathic PCP:*
   - **Long-acting** GnRH agonists will suppress pituitary FSH & LH and ovarian estrogen (delaying progression into puberty).
   - Treatment is continued until reaching appropriate age for resumption of pubertal development.
2. *Surgical removal of functioning ovarian tumors.*
3. *Primary hypothyroidism is treated with thyroid replacement therapy (Eltroxin).*
4. *Adrenal tumors are treated with surgery or irradiation.*

Menopause

1. Define menopause & mention remote health hazards (sep 2009)

Definition:

The final menstruation that occur at the end of climacteric. It is defined as 12 months of amenorrhea after the final menstrual period, in absence of any other pathological or physiological cause.

Remote health hazards:

1. *Cardio vascular system changes*

   Pathogenesis:

   Estrogen deficiency may lead to hypercholesterolemia with increased LDL and decreased HDL.

   Clinical manifestation:

   Reversed LDL/HDL ratio may predispose to ischemic heart disease myocardial infarction, atherosclerosis, hypertension, and stroke.

2. *Osteoporosis:*

   Osteoporosis is a disorder characterized by gradual decreased bone mineral density leading to compromised bone strength with increased risk of bone fracture.

   Pathogenesis:

   Estrogen deficiency results in accelerated bone mineral calcium loss and increase activity of osteoclasts, affecting mainly the vertebrae, femoral neck, distal radius and calcaneus.

   Clinical manifestations:

   Bone demineralization is usually a silent disease that manifests years after menopause with decreased height, increased curvature of spine, silent fractures of the vertebrae or fractures of the hip and long bones on exposure to mild trauma.

   Risk factors:

   Premature menopause, heavy smoking, lack of exercise, low body weight, together with hereditary and genetic factors (white race).

   Diagnosis:

   X-ray bone densitometry (DEXA).

   Prevention:

   1. Diet with rich calcium 1500 mg daily.
   2. Vitamin D 600-800 IU/day.
   3. Healthy life style; including weight bearing exercises, stop smoking and avoid long term corticosteroid therapy.

   Treatment:

   1. Drugs that slow bone breakdown during bone remodeling:
      - Bisphosphate: orally decrease non-vertebral fractures.
      - Calcitonin: nasal spray increase the vertebral bone mass and reduce fracture risk.
   2. Drugs that have anabolic effect that stimulate bone remodeling:
      - Teriparatide: IM decrees vertebral and non-vertebral fracture.
   3. Hormone therapy:
      - Although effective in prevention and management of osteoporosis, is rarely used as primary therapy. Except in cases which need HT for control of flushes or in cases of

premature menopause

- Selective Oestrogen Receptor Modulators (SERM): raloxifene has a combined oestrogen effect on bone and anti-oestrogen effect on the breasts and uterus. It is approved for prevention of osteoporosis but may induce hot flashes.
- Phyto-oestrogen: Plant substitutes have a weak oestrogen action.

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2. Menopause; mention definition, types, endocrine changes, and remote health hazards. (june 2010)

3. Endocrine changes of menopause (june 2012)

Definition:

The final menstruation that occur at the end of climacteric. It is defined as 12 months of amenorrhea after the final menstrual period, in absence of any other pathological or physiological cause.

Types:

1. *Natural menopause:*
   - Occurs due to intrinsic ovarian failure that usually occurs between 45-55 years with a median age of **51** years.
   - It is characterized by complete or near complete, ovarian follicular depletion with subsequent cessation of ovarian estrogen (E2) secretion.
     - Late or delayed menopause: menopause occurring > 55years of age.
     - Early or premature menopause: occurring between 40-45 years of age
2. *Induced menopause:*

   Menopause can be artificially induced:

   a. Surgically: as after bilateral oophorectomy.
   b. Ablation of ovarian function; as pelvic irradiation or systemic chemotherapy.
   c. Medically: during the use of long acting GnRH agonists in the management of endometriosis.
3. *Premature menopause:*

   Menopause occurring between 40-45 years of age. May be either:

   a. <u>Premature ovarian insufficiency (POI):</u>

   Due to congenitally deficient number of ovarian follicles at puberty, leading to their early exhaustion at relatively young age.
   - Idiopathic: no underlying etiology.
   - Gonadal dysgenesis: as in cases of mosaic turner syndrome (46 XX/45 XO)

   b. <u>Induced:</u>

   Surgical, irradiation or chemotherapy before the age of 45 years.

Endocrinal changes:

- Decreased serum inhibin-B levels that started at the perimenopause.
- Marked and persistent Decrease in ovarian E_2 levels.
- Marked Increase in serum (FSH), (LH) level.
- Decrease in sex hormone binding globulin (SHBG).
- Increased frees testosterone levels.
- Persistent production of ovarian testosterone.

Remote health hazards:

See before.

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4. Clinical features of menopause (june 2012)

Most menopausal manifestations are secondary to chronic low serum E2 levels.

Symptoms usually start gradually in the perimenopause, increase sharply after FMP and extend years into menopause.

Menstruation may stop abruptly, however the FMP is more commonly preceded by a period of oligomenorrhea in the late premenstrual period.

1. *Vasomotor: Hot Flushes & cold sweating:*
   - Hot flushes: are recurrent waves of heat over the chest, neck, and face followed by cold sweating. A flush may last for 1-5 minutes. and may be associated by Palpitation, headache, and dizziness.
   - Flushes affect at least 50% of menopausal women but with variable degrees.
   - Start in perimenopause and become more aggressive in the menopause.
   - Result from inappropriate stimulation of the thermoregulatory centres in the hypothalamus with vasodilatation of the skin over chest, neck, and face causing skin temperature rise although core body temperature does not change.
2. *Nervous and psychological:*

   Anxiety, irritability, mood changes, lack of concentration, insomnia.
3. *Gastro-intestinal:*

   Constipation, abdominal distension, tendency to weight gain.
4. *Urinary:*

   Frequency, dysuria, stress incontinence with predisposition for urinary tract infections.
5. *Dyspareunia:*

   As vaginal atrophy, dryness and senile vaginitis.
6. *Tendency toward pelvic organ prolapse:*

   Uterine and vaginal prolapse due to weakness and atrophy of pelvic and cervical ligaments.
7. *Androgenic effects:*

   Increased facial hair and baldness.
8. *Symptoms related to osteopenia and osteoporosis:*

   Decreased height, increased curvature of the spine, silent fracture of the vertebrae, fracture of hip and long bones on exposure to mild trauma.

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5. Menopausal osteoporosis; etiology diagnosis & treatment. (june 2013)

See before.

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6. Hormone replacement therapy in menopause: benefits and risks, indication and contraindications, types, schedule, route of administration and follow up (june 2011)

Benefits of HRT:

1. It reduces menopausal symptoms as hot flushes, insomnia and mood disorder.
2. Treats vaginal dryness and atrophy which cause dyspareunia and senile vaginitis
3. Prevents or reduce the risk of osteoporosis during the period of the therapy.

Risks of HRT:

1. Small but significant increase for CVS disease, stroke, venous thromboembolism.
2. Small but significant increase in risk of the breast cancer.
3. Endometrial cancer risk if used for more than 5 years (relative risk 1.3).

Indications of HRT:

1. Menopausal symptoms affecting the patient life style and psychological conditions.
2. Premature menopause (idiopathic or surgically induced) until the age of natural menopause.
3. To prevent osteoporosis in high risk cases. Beneficial effect occur only during treatment and stop with cessation of treatment.

Contraindications:

1. Undiagnosed abnormal bleeding from genital tract.
2. Known or suspected breast cancer or oestrogen dependent neoplasia.
3. History of DVT, stroke, or thromboembolic embolism
4. Active liver disease.

Types of hormone used:

- Oestrogen + progestin therapy: where progestin is added to avoid oestrogen induced endometrial hyperplasia and endometrial carcinoma.
- Oestrogen only therapy: suitable for women have undergone hysterectomy (no risk of oestrogen induced endometrial hyperplasia and endometrial carcinoma).
- Natural oestrogens are preferred over synthetic ones: conjugated equine oestrogen or oestradiol valerate.
- Synthetic Gestagens are preferred being more effective in smaller dose. side effects include: mastalgia, mood changes, PMS like syndrome, weight gain.....

Schedule and route of administration:

- Continuous combined E/ PRG therapy: daily oral tablets throughout duration of therapy (1-2 years). No withdrawal bleeding expected.
- Cyclic combined E/PRG therapy: daily oestrogen tablets for 3 weeks, PRG added last 12 days. Treatment stopped for one week in which withdrawal bleeding is expected.
- Oestrogen only therapy: daily oral doses, sub dermal implants, or trans derma l patches are used for women who have undergone hysterectomy.
- Oestrogen vaginal cream preparation: for local application in cases of vaginal atrophy.

Follow up for HRT cases:

- Periodic mammography every 1-2 year.
- Pap smear yearly
- Bone densitometry
- Ultrasonography for cases with bleeding
- Endometrial biopsy for abnormal bleeding pattern to avoid delay in the diagnosis of endometrial carcinoma.

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7. Hormones used for treatment of menopause: Enumerate, indication and contraindication. (sep.2009,2011)

See before.

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8. Changes in body systems associated with menopause.

- Changes start in the climacteric period and continue gradually and persistanly after cessation of menstruation
- They are almost related to androgen deficiency.

1. *The vagina:*

   Becomes smaller thinner, with gradual loss of its rugae, decreased vascularity, and increased vaginal PH. Vaginal smears become atrophic.

2. *The pelvic ligaments:*

   Become weaker predisposing to pelvic organ prolapse

3. *The uterus:*

   Become smaller in size and myomas under go atrophy.

4. *The endometrium:*

   Becomes thin and atrophic (<5mm)

5. *The cervix:*

   Becomes gradually flushed with vaginal fornices, and squmo-cloumnar junction migrates higher in the endocervical canal.

6. *The urethra and bladder mucosa:*

   Show loss of elasticity, bladder dysfunction, and stress incontinence(due to relaxation of pelvic ligaments)

7. *The breasts:*

   Become gradually smaller and flabby with progressive fatty replacement of breast tissue and atrophy of active glandular elements.

8. *The skin:*

   Gradual decrease in thickness and collagen content.

9. *Increased facial hair and androgenic alopecia*
10. *Gradual change in cognitive function and mood swings*
11. *Decreased mineral bone density*
12. *Nervous and psychological changes*

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9. Management of the menopause.

- Reassurance about the physiological nature of symptoms
- Education of healthy life style: calcium rich diet, suitable exercise, and avoid smoking
- Symptomatic treatment for some symptoms: sedatives or anti depressants
- Periodic tests for early detection of premalignant and malignant lesions including: mammography, TVS, pap smear, cytology, colposcopy.
- Prevention and treatment of osteoporosis
- + HRT.

Amenorrhea

1. **Discuss outflow tract disorders as cause of amenorrhea.**
   Discuss clinical picture and treatment of imperforate hymen.

- Outflow tract disorders are associated with obstruction of menstrual flow at the level of hymen, vagina, or cervix.
- This leads to **primary amenorrhea** (usually at young age), with **normal development of secondary sexual characters.**
- This type is called "**false amenorrhea**" or "**cryptomenorrhea**"; because menstruation occurs but it is not revealed.

They include:

1. Imperforate hymen.
2. Transverse vaginal septum.
3. Cervical atresia.

Imperforate hymen

Definition:

- Congenital anomaly, in which the orifice of the hymen is absent.
- The **commonest cause of cryptomenorrhea** (affects 0.1% of newly born females).

Pathogenesis:

Menstruation will start at puberty, but blood will accumulate behind the imperforate hymen, leading to:

- Hematocolpos → blood accumulating in vagina (mainly).
- Hematometra → blood will extend to uterine cavity in longstanding cases.
- Hematosalpinx → blood will collect in the fallopian tubes (retrograde from uterus).

Clinical presentation:

Young girls with primary amenorrhea + well-developed secondary sexual characters.

Symptoms:

1. Lower abdominal pain synchronous with the time of the menstrual period.
2. Acute urine retention.

Signs:

A. *General:*
   2ry sexual characters are well developed
B. *Abdominal:*
   May show suprapubic bulge only if hematocolpos is large enough to extend upwards to suprapubic region
C. *Local:*
   1. The orifice of hymen is absent.
   2. If the hematocolpos is large enough, the hymen will bulge with a slight bluish color.
   3. Sizable hematocolpos can be easily palpated (PR examination)

Investigation:

Pelvic ultrasound is the gold standard in diagnosis.

Treatment:

1. Surgical cruciate incision to divide the imperforate hymen (hymenotomy).

2. Certificate of virginity
3. Catheterization for urine retention

Transverse vaginal septum

Definition:

- One or more transverse vaginal septae maybe congenitally present at any level between the hymeneal ring and the cervix.
- This leads to occlusion of lower, middle, or upper segment of the vagina.
- Rare condition, but is the **2nd most common cause of cryptomenorrhea.**

Clinical presentation:

Cryptomenorrhea as seen with imperforate hymen.

Diagnosis:

- Hematocolpos diagnosed by US.
- Hymen is normal.

Treatment:

Surgical excision of the transverse septum.

Cervical atresia

Definition:

A very rare condition in which there is failure of canalization of cervical canal as part of Mullerian dysgenesis.

Clinical presentation:

Cryptomenorrhea as seen with imperforate hymen.

Diagnosis:

Pelvic US shows marked hematometra, absent cervix, and no hematocolpos.

Treatment:

- Difficult and controversial.
- Attempts of cervical reconstruction and canalization, with poor results.
- If fails, hysterectomy maybe the only option.

---

2. Mention 3 criteria and 3 investigations to diagnose Turner syndrome.

Definition:

Turner syndrome (gonadal dysgenesis): a chromosomal defect in which one X chromosome is missing (45, XO) and ovaries are replaced by fibrous tissue (streak gonads).

Incidence:

The **commonest cause of primary amenorrhea** (30% of cases).

Clinical presentation:

1. Primary amenorrhea.
2. External phenotypic characters of Turner syndrome, as:
    - Low hairline.
    - Short stature.
    - Webbing of neck.
    - Increased carrying angle at the elbow.
    - Cardiac anomalies, as coarctation of aorta.

Investigations:

1. Pelvic US to confirm small uterus and streak gonads.

2. Hormonal profile for FSH, LH, E2 and serum androgens.
3. Karyotyping by a blood sample to diagnose 45, XO pattern and exclude mosaic Turner.
4. ECG and ECHO for cardiac abnormalities.

Treatment:

Hormone replacement therapy (HRT) in the form of cyclic combined EST/PRG (21 days therapy and 1 week free) to:

- Induce regular cycles.
- Preserve secondary sexual characters.
- Prophylactic against premature menopause (osteoporosis, ..).

NB:

- In some cases with **mosaic karyotype** (45, XO/46, XX), spontaneous menstruation and pregnancy may occur (2-5%).
- But most cases will develop premature ovarian failure (POF) and premature menopause.

---

3. Causes and diagnosis of secondary amenorrhea.

Definition:

Cessation of already present menstruation more than 3 successive cycles.

Causes:

1. (Pregnancy is the commonest cause of secondary amenorrhea "physiological").
2. **Polycystic ovary (PCO):**
   Commonest cause of secondary amenorrhea due to pathologic anovulation.
   Details: see anovulation chapter.
3. **Hyperprolactinemia:**
   Details: see later.
4. **Endocrine disorders:**
   Some endocrine disorders maybe severe enough to cause hypothalamic-pituitary-ovarian disturbance, leading to transient secondary amenorrhea.
   Hypothyroidism:
   - Elevated TRH results in elevated serum PRL.

   Cushing syndrome:
   - Increased adrenal activity leads to hyperandrogenic state.

   Management:
   - Correction of the endocrine disorder, e.g. Eltroxin for hypothyroidism, Thiouracil for hyperthyroidism, ...
5. **Drug-induced amenorrhea:**
   A. *GnRH agonists:*
      - **Long-acting** GnRH agonists will induce **initial stimulation** of GnRH for short period, followed by **longer suppression** of FSH & LH due to down-regulation of receptors → secondary amenorrhea.
      - Useful in treatment of severe endometriosis, severe AUB, and to reduce the size of large uterine myomas prior to surgery if necessary.

   B. *Progestins:*
      - **Continuous** synthetic progesterone therapy will prevent endometrial shedding and inhibit GnRH pulses → secondary amenorrhea.

- Normal menses is resumed after stopping therapy.

C. *Combined EST/PRG therapy:*
- **Continuous therapy** will also prevent endometrial shedding and inhibit GnRH pulses as long as therapy continues.
- **Post-pill amenorrhea** may occur in 1% of women after long use of combined OCPs due to chronic decrease in gonadotropin levels.
- The condition is self-limiting. Normal menses is spontaneously resumed in 2-6 months.

D. *Androgenic drugs:*
- As Danazol (previously used in treatment of endometriosis) induces atrophic endometrial changes by its androgenic and progestational effects.

E. *Anti-psychotics and tricyclic anti-depressants:*
- Stimulate prolactin secretion (anti-dopaminergic drugs).

6. **Rapid weight loss and excessive exercise:**

A minimum of 20% body fat by weight is required for initiation of menarche and maintenance of menses.

7. **Excessive stressful events:**

Usually associated with increased circulating endorphins and may cause 2ry amenorrhea in female athletes as marathon runners, ballet dancers, etc. esp. those with markedly decreased body fat.

8. **Ashermann's syndrome (intra-uterine synechiae):**

Definition:

Acquired intra-uterine adhesions which prevent endometrial proliferation → secondary amenorrhea.

Etiology:

1. Iatrogenic: vigorous curettage during D & C procedures esp. with surgical evacuation (**commonest cause**).
2. Endometritis: post-abortive, puerperal, IUD-induced, or TB.

Clinical presentation:

2ry amenorrhea following endometritis or curettage.

Diagnosis:

Hysterosalpingography (HSG) and hysteroscopy are excellent diagnostic tools.

Treatment:

- Lysis of adhesions best under vision via hysteroscopy, or through D & C like procedure.
- Followed by cyclic combined EST/PRG therapy for at least 3 cycles to restore endometrial regeneration.
- Very bad prognosis and recurrence is common.

9. **Premature ovarian failure (POF):**

Definition:

Early exhaustion of ovarian primordial follicles before the age of 40 years leading to premature menopause.

Etiology:

1. Idiopathic (associated with autoimmune ovarian destruction).
2. Karyotype abnormalities as loss of a small portion of X chromosome.

3. Viral infections as mumps.
4. Induced by radiation therapy, chemotherapy, or surgical removal.

Clinical presentation:

2ry amenorrhea with high FSH & LH levels (premature menopause).

Treatment:

HRT (combined EST/PRG) to avoid complications of premature menopause.

Resistant ovary syndrome:

- Viable ovarian follicles fail to respond to pituitary gonadotropins (FSH & LH) due to defect in receptors.
- 2ry amenorrhea occurs with high FSH & LH levels.
- In few cases, the condition is temporary (some may ovulate & conceive).

10. Anterior-pituitary gland disorders:

A. Pituitary adenoams: (see hyperprolactinemia)

Hyper-prolactinemia itself is responsible for 20% of cases of 2ry amenorrhea (suppression of GnRH and LH surge leading to a state of chronic oligo-ovulation, anovulation and finally 2ry amenorrhea).

Prolactinomas:

- The commonest cause of hyperprolactinemia.
- The most common pituitary cause of 2ry amenorrhea.

Histological classification:

- Microadenoma (< 10 mm in size):
  - The commonest.
  - Moderate elevation in serum PRL.
- Macroadenoma (> 10 mm in size):
  - Relatively rare.
  - High serum PRL and signs of increased intracranial tension.

Diagnosis and treatment:

See hyperprolactinemia.

B. Empty-sella syndrome:

Definition:

Enlarged sella turcica that is not entirely filled with pituitary tissue.

Pathogenesis:

Defect in diaphragma sella that allows CSF pressure to enlarge the sella (pressure atrophy of pituitary gland).

Clinical presentation:

2ry amenorrhea or oligohypomenorrhea with hyperprolactinemia.

Diagnosis:

MRI or CT scan is the main diagnostic tool.

C. Pituitary insufficiency:

Diminished FSH and LH secretions leading to a state of chronic anovulation with hypogonadtrophic amenorrhea.

Causes:

Rare and include:

1. Sheehan's syndrome:
   Anterior pituitary necrosis following severe postpartum hemorrhage.
2. Simmond's disease.
   Necrosis due to any other cause.
3. Radiation necrosis, pituitary infarctions, and non-lactotrophic adenoma.
4. Infiltrative lesions of pituitary gland, as lymphocytic hypophysitis.

Management:

- According to the cause, but generally HRT with cyclic combined EST/PRG.
- Induction of ovulation can be attempted using IM FSH/hCG preparations.
- Clomiphene citrate is contraindicated due to inability of pituitary to produce FSH & LH.

11. Hypothalamic and CNS disorders:

Decreased GnRH pulse frequency leading to low FSH & LH secretion, absent LH surge, low E2 level, and chronic anovulation (hypogonadotrophic amenorrhea).

A. Psychiatric disorders:

i. Emotionally stressful events:

- As family problems, work, study, travel, death, or severe illness.

ii. Pseudocyesis (false pregnancy):

- Rare condition that may occur in women with extreme desire of pregnancy.
- Unknown etiology but maybe due to **voluntary** alteration of hypothalamic function.
- Prolactin levels maybe elevated enough to cause galactorrhea.

Anorexia nervosa:

- May affect 1% of women.
- Bulimia (induced vomiting) maybe present in 50% of cases.

iii. Rapid weight loss and excessive exercise:

See before.

B. Drug-induced amenorrhea:

See before.

C. Hypothalamic tumors and infiltrative lesions (rare):

i. Craniopharyngioma:

Associated with visual field defects and calcification in x-ray.

ii. Lymphoma, Langerhans cell histiocytosis, and sarcoidosis:

Decreased GnRH secretion, low or normal serum FSH and LH, and amenorrhea.

NB:

Most women will have 2ry amenorrhea with one or more neurologic symptoms as severe headache, change in personality or marked mood changes.

Assessment of 2ry amenorrhea:

"See scheme page 5"

i. *Exclude pregnancy:*

By Ultrasound or pregnancy test.

ii. *Exclude PCO:*

1. History of preceding oligohypomenorrhea.
2. Examination for evidence of hirsutism (face, in between breasts, inner thighs, and back).
3. Examination for obesity (weight, height & BMI).
4. History of infertility.
5. Hormonal assay (FSH, LH, PRG, Testosterone, FSH/LH ratio, serum insulin).
6. Pelvic US (TAS/TVS): showing increased volume, dense stroma, and peripheral follicles (necklace or string of pearls appearance).

   ** TVS: Trans-Vaginal Scan.

   ** TAS: Trans-Abdominal Scan.

iii. *Exclude hyperprolactinemia:*

1. History of preceding irregular cycles and oligomenorrhea.
2. Symptoms of increased ICT (headache, projectile vomiting, blurred vision).
3. Breast examination for galactorrhea.
4. Hormonal assay for serum PRL, TRH & PRG.
5. MRI or CT scan on brain for prolactinoma, empty-sella & craniopharyngioma.

iv. *Exclude thyroid disorders:*

1. Clinical examination.
2. Serum TRH, TSH, T_3, T_4, anti-thyroglobulin antibodies.

v. *Exclude drug-induced amenorrhea:*

1. History of drug intake (OCPs, GnRH, Danazol, Visanne, antidepressants & GIT drugs).
2. History of medical illness.

vi. *Exclude psychiatric disorders:*

Stressful exercise, rapid weight loss, depression & mood swings.

vii. *Exclude functioning ovarian tumors:*

1. Feminizing tumors: breast development, irregular uterine bleeding.
2. Virilizing tumors: cliteromegaly, hirsutism, deepening of voice, and increased muscle bulk.
3. Abnormal E2 or Testosterone levels.
4. Pelvic US revealing solid adnexal mass (ovarian tumor).

viii. *Exclude Ashermann syndrome:*

1. History suggestive of endometritis (post-abortive, puerperal, IUD-induced, TB, ..).
2. TVS; shows absent trilaminar endometrial plate.
3. HSG; shows obliterated cavity (strongly suggestive).
4. Hysteroscopy is conclusive and therapeutic.

ix. *Exclude POF & POI:*

1. Menopausal symptoms as hot flushes, irritability, weight gain, insomnia, ..
2. Hormonal assay showing elevated FSH & LH with low E2 and poor AMH.
3. TVS shows menopausal uterus, atrophic endometrium, small ovaries with inactive follicles.

** POF = Premature Ovarian Failure.

** POI = Primary Ovarian Insufficiency.

---

4. Surgical management in amenorrhea.

Surgical correction maybe required to **relieve obstruction** or to **allow for coital function.**

i. Hymenotomy:

- Cruciate incision for imperforate hymen.

ii. Excision of transverse vaginal septum:

- In case of cryptomenorrhea.

iii. Re-construction of a functional neo-vagina with skin grafting "McIndoe procedure":

- In case of vaginal agenesis.
- Also done for cases with testicular feminization after marriage.

iv. Gonadectomy:

- In patients with Y containing gonad.
- Gonadectomy should be done once diagnosis is confirmed due to risk of malignant transformation into gonadoblastoma.
- However in testicular feminization syndrome, gonadectomy is better postponed after completion of puberty to allow normal growth.
- Such cases will require lifelong HRT to improve sexual characters and protect against premature menopause.

v. Pituitary macroadenoma:

- Rarely, cases with increased ICT or those unresponsive to medical treatment may require trans-sphenoid surgery or gamma knife.

vi. Laparoscopic Ovarian Drilling (LOD):

- In cases of PCO resistant to induction of ovulation by medical treatment.

---

5. Hyperprolactinemia (causes).

Galactorrhea (definition, causes & treatment).

Prolactin:

- Polypeptide hormone secreted by lactotrophic cells of anterior pituitary gland.
- Secretion is controlled by hypothalamic **prolactin inhibiting factor (PIF)** known as **dopamine**.
- Prolactin is responsible for initiation and maintenance of lactation in females.

Hyperprolactinemia:

- Elevated serum prolactin levels (N: 2.9-29 ng/ml).

Galactorrhea:

- Continuous extrusion of milk from nipples in absence of recent pregnancy or lactation.
- Almost always secondary to hyperprolactinemia.

Etiology:

1. *Physiologic causes:*

   During pregnancy and lactation.

2. *Drug-induced:*

   <u>These drugs act by reduction of hypothalamic secretion of dopamine (PIF), and include:</u>

   - Phenothiazine derivatives.
   - Reserpine.
   - Psychotropic drugs.
   - Metoclopramide.
   - Estrogen.
3. *Primary hypothyroidism:*

   Due to persistently elevated TRH levels.
4. *Prolactin-secreting pituitary adenoma (Prolactinoma):*

- Microadenomas (< 10 mm): common cause of hyperprolactinemia.
- Macroadenomas (> 10 mm): rare, maybe associated with signs of increased ICT (headache, projectile vomiting, blurred vision, ..).

5. *Hypothalamic disorders:*

- Severe stress and psychological conditions.
- Hypothalamic tumors (craniopharyngioma): cause damage to hypothalamus, or compression on pituitary stalk interfering with production of prolactin or transport of dopamine.

Clinical picture:

3M (<u>M</u>astalgia/**<u>M</u>**enstrual disorders/مابتولدش)

<u>One or more of the following:</u>

1. <u>Mastalgia</u> (breast pain and tenderness), with or without galactorrhea.
2. <u>Menstrual disorders</u> (irregular cycles and 2ry amenorrhea): due to chronic anovulation.
3. <u>Infertility</u>: due to anovulatory dysfunction (PRL interferes with GnRH pulses and with ovarian sensitivity to pituitary gonadotropins).

Diagnosis:

1. *History:*

   Breast pain (mastalgia) and breast milky secretions (galactorrhea), with or without menstrual disturbance or infertility.
2. *Clinical examination:*

   Gentle pressure on nipples results in extrusion of milky secretion.
3. *Investigations:*

   <u>Laboratory:</u>

   - Elevated serum PRL.
   - Marked elevation > 100 ng/ml suggest PRL secreting adenoma.

   <u>CT scan and MRI:</u>

   - In case of persistently high serum PRL or in case of signs of increased ICT (suggestive of brain tumor).

Treatment:

1. Stop medications that may cause hyperprolactinemia.
2. Treat primary hypothyroidism by thyroid hormone replacement therapy (as Eltroxin).
3. <u>Drugs used for treatment of hyperprolactinemia (dopamine agonists):</u>
   - Bromo-ergo-cryptine: 2 mg (1-2 tablets daily, until normal PRL levels restored).
   - Lisuride hydrogen maleate: 0.2 mg (1-2 tablets daily, until normal PRL levels restored).

- Cabergoline: 0.5 mg (1/2 tablet twice weekly for 4 weeks, until normal PRL levels restored).

4. Treatment of pituitary adenoma:
   - Medical treatment, using dopamine agonists is the primary treatment for most cases.
   - Trans-sphenoidal surgery or Gamma knife in case of failure of response to medical treatment, or macroadenomas with CNS pressure symptoms.

Other topics:

1.

	Complete or partial Mullerian agenesis "Mayer Rokitansky Kauster Hausser syndrome"	**Complete androgen insensitivity syndrome (CAIS) "Testicular feminization syndrome"**
Definition	Females (46, XX) with congenital genetic defect resulting in failure of development of Mullerian structures (uterus, cervix & upper vagina).	Males (46, XY) with X-linked recessive disorder resulting in defect in peripheral androgen receptors (androgen resistance).
Incidence	2nd most common cause of 1ry amenorrhea (20%, after Turner syndrome). 1:4000 females. Commonly associated with skeletal & urinary tract anomalies.	Rare, but 3rd most common cause of 1ry amenorrhea after Mullerian agenesis.
Clinical presentation & diagnosis	1ry amenorrhea with normal 2ry sexual characters. Normal ovaries (develop from genital ridge) & normal production of estrogen (intact hypothalamic-pituitary-ovarian axis). PV examination (if not virgin): blind-ended short vagina, with absent cervix and uterus. Pelvic US: absent uterus and upper vagina with normal ovaries. Karyotyping: blood sample for genetic study (46, XX).	Failure to develop testosterone-dependent male sexual characters inspite of normal testosterone. So they develop phenotypically as females. 1ry amenorrhea, female external genitalia, and a blind-ended vagina.
Pathogenesis	Unwanted intra-uterine exposure to anti-Mullerian hormone (AMH), with failed development of Mullerian duct.	Male gonadal tissue maybe present in labia or inguinal canal, but incapable of spermatogenesis. The presence of Y chromosome, and Mullerian inhibiting factor (MIF) leads to failure of Mullerian system development (uterus, cervix and upper 1/3 vagina).
Management	Short vagina can be managed surgically by lengthening the vaginal canal through vaginoplasty	- Gonadectomy must be performed (20% risk of gonadoblastoma) after puberty (to allow growth and

	(McIndoe procedure).	development of 2ry sexual characters). - Creation of a neo-vagina surgically via McIndoe procedure, if the patient requires, as such cases are reared as females. - HRT in the form of estrogen preparation to keep external female appearance & prevent premature menopause.

2. Congenital GnRH deficiency.

- Rare condition that presents with 1ry amenorrhea + infantile sexual development (hypo-gonadotrophic hypogonadism).
- Kallmann's syndrome: congenital GnRH deficiency + anosmia due to congenital failure of neuronal migration of olfactory placode in the nose.
- <u>Treatment:</u>

– HRT with combined EST/PRG therapy to help preserve 2ry sexual characters, uterine size & attempt to induce uterine withdrawal bleeding.

– In cases with normal-sized uterus and responsive endometrium, induction of ovulation maybe tried using FSH/LH/hCG protocols.

3.

Common causes of 1ry amenorrhea	Common causes of 2ry amenorrhea
Cryptomenorrhea (imperforate hymen). Turner syndrome (45, XO). Mullerian agenesis (46, XX). Androgen insensitivity (46, XY). Congenital GnRH deficiency. Kallmann's syndrome.	PCO. Hyperprolactinemia. Endocrine disorders (hypothyroidism). Drug induced (post-pill – GnRH agonists). Rapid weight loss and excessive exercise. Ashermann's syndrome. POF – POI.

4. Clinical assessment of amenorrhea.

Clinical assessment:

- Evaluation of cases of Amenorrhea will start by:
  - Thorough history.
  - General examination.
  - Local examination (inspection, palpation, PV & bimanual Examination).
- A preliminary diagnosis may be reached at this step which will be confirmed by (special investigation) including primarily pelvic US & hormonal assay.
- Further investigations as other lab tests, Endoscopy, CT or MRI may be required to confirm the suspected diagnosis.

* 1st → Differentiate between physiologic & pathologic amenorhea by:

- Confirming puberty.

- Excluding → pregnancy, lactation & menopause.

* 2nd → differentiate between 1ry & 2ry amenorrhea by History, especially menstrual history.

A) In case of 1ry Amenorrhea:

1- Exclude cryptomenorrhea:

- Patient's age & signs of pubertal development.
- Inspection of external genitalia for imperforate hymen.
- PR examination (if PV couldn't be performed) to palpate hematocolpos.
- Pelvic US to confirm or exclude presence of hematocolpos, hematometra and to localize the site of outflow tract obstruction.

2- Exclude turner syndrome (gonadal dysgenesis) through:

- Examination of external phenotypic features → short stature , webbing of neck, …
- Cardiac examination, ECG, and Echocardiography, for cardiac abnormality.
- Pelvic US → to discover small uterus & streak gonads.
- Hormonal profile → FSH, LH, E2 & serum androgens.
- Karyotyping by a blood sample to diagnosis 45, XO and exclude mosaic turner.

3- Differentiate between Mullerian agenesis & complete androgen sensitivity syndromes:

	Mullerian agenesis	**Complete androgen insensitivity syndrome (CAIS)**
Karyotype	XX	XY
Gonads	Ovaries	Testicles (inguinal)
Uterus	Absent	Absent
Vagina	Short vaginal pouch	Absent/small dimple
Axillary/ pubic hair	Present	Absent/sparse
Associated anomalies	Renal, skeletal & deafness	Absent
Reproduction	Possible oocyte retrieval	Not possible
Surgical interventions	Vaginoplasty only if short pouch	Vaginoplasty & gonadectomy
EST HRT	Not required	Required for appearance & osteoporosis

B) In cases of 2ry Amenorrhea:

See before.

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5. Investigations of a case of primary amenorrhea.

Definition:

Menstruation has never started in the female:

- Till the age of 14 years: without development of 2ry sexual characters.
- Till the age of 16 years: regardless development of 2ry sexual characters.

Investigations:

Discuss according to causes.

Disorders of menstrual cycle
[Dysmenorrhea & AUB]

1. **Clinical types of dysmenorrhea.**
2. **Enumerate different types of dysmenorrhea and their causes.**
3. **Define spasmodic dysmenorrhea, mention aetiology, diagnosis and ttt.**

Definition:

Dysmenorrhea is pain and cramping during menstruation that interfere with normal activities and requires the use of medications to control the symptoms. Pain may range from mild discomfort to severe pain. That causes some patients to be bedridden for 1-3 days\month.

Dysmenorrhea can be classified into:

1. 1ry dysmenorrhea (Spasmodic type)
2. 2ry dysmenorrhea (Congestive type)

	Primary dysmenorrhea (Spasmodic)	**Secondary dysmenorrhea (Congestive)**
Definition	*Idiopathic menstruation pain without identification pathology. *It is always associated with **ovulatory** cycles, with no obvious organic cause.	*It is painful menses due to underlying pathology (fibrosis, endometriosis, adenomyosis, PID, cervical stenosis) *It may occur in both **ovulatory** and **anovulatory** cycles.
Etiology	1. Increase level of endometrial prostaglandins (PGLs) 2. Additional psychological component may be involved in some patients. 3. The condition usually spontaneously improves after term pregnancies and deliveries.	• Symptoms are secondary to identifiable cause.
Diagnosis	• **Age**: usually in younger women < 20 years of age • **Pain**: on the first and second days of ovulatory cycles. • **Associated symptoms**: nausea, vomiting and headache. • **Physical examination**: No obvious abnormalities or pelvic pathology. • **Pelvic US**: normal uterus, ovaries and adnexa.	• **Age**: commonly mid-aged female (30-35) years >> parous and null-parous • **Pain**: starts few days before menses and gradually decreases by onset of menses. It is dull aching, colicky, supra-pubic pain **commonly associated with** low backache that usually occurs after several years of painless menstruation. • **Physical examination**: may reveal sizable uterine and

		adrenal masses. • **Pelvic US**: may diagnose smaller uterine myomas, ovarian endometriosis and tubo-ovarian masses. • **Laparoscopy**: for diagnosis of PID, endometriosis and pelvic adhesions.
Treatment	1. **Reassurance:** The first and most important step. 2. **NSAIDs:** The first and most important line of treatment (aspirin, ibuprofen, ketoprofen and naproxen) they are taken at the onset of menses, continued for 1-3 days then taken if needed 3. **Combined oral contraceptive pills (OCPs):** The second line of ttt in cases which do not get adequate pain relief with the use of NSAIDs alone, or cannot tolerate their side effects.	1. **Medical ttt:** To control pain via NSAIDs 2. **Hormonal ttt:** OCPs, gestagens and GnRH agonists 3. **Surgical ttt:** For larger myomas, endometriomas and tubo-ovarian mass.

4. Clinical patterns of abnormal bleeding. "Just enumerate"

1. *Menorrhagia:*

Excessive menstrual bleeding

2. *Metrorrhagia:*

Significant intermenstrual bleeding

3. *Menometrorrhagia:*

Heavy bleeding both during menstruation and in the intermenstrual period. The cycle becomes irregular.

4. *Polymenorrhagia:*

Frequent menstrual cycles that occur <21 days.

5. *Oligomenorrhea:*

*Infrequent menstrual cycles that occur >35 days.

*Secondary amenorrhea is diagnosed when no menstruation occurs for >6 months

6. Hypomenorrhea:

Scanty menstrual bleeding

6. *Post-menopausal bleeding (PMB):*

Vaginal bleeding in menopausal women

7. *Contact bleeding (CB):*

Bleeding on touching the cervix

---

5. Discuss shortly the different types of DUB and give short account on metropathia hemorrhagica.

6. Metropathia hemorrhagica: mention definition, aetiology, ttt.

DUB

Definition:

It is AUB that occurs in absence of recognizable pelvic pathology, medications, systemic disease, blood dyscrasias, trauma or pregnancy. It is diagnosis of exclusion.

Incidence:

60% of AUB

Etiology:

Chronic anovulation leading to alteration in endometrial vascular homeostasis

Pathophysiology:

a. In ovulatory cycles: menstruation occurs at the end of luteal phase due to withdrawal of progesterone secondary to corpus luteum dysfunction. This leads to secretion of prostaglandin F2 alpha leading to vasoconstriction and ischemia of endometrium , shedding of superficial and intermediate layers of endometrium.
b. In anovulatory cycles: proliferative endometrium outgrows its blood supply leading to irregular bleeding. The less amount of prostaglandin F2 alpha makes the endometrium less efficient in controlling amount and duration of bleeding
c. Chronic anovulation: prolonged estrogenic effect on endometrium unopposed by progesterone as in PCOS leading to endometrial hyperplasia and later on endometrial carcinoma.

Types:

1. Clinical:
   a. Cyclic (usually ovulatory)
   b. Acyclic (usually anovulatory)
2. Histopathological:
   a. Ovulatory (usually cyclic)
   b. Anovulatory (usually acyclic)

Metropathia hemorrhagica

- **Definition:**
  - Excess volume of blood of irregular unexpected intervals from abnormal pathological hyperplastic polypoid endometrium due to persistent follicular phase.
  - Follicle does not rupture leading to inc. E2 with inc. proliferative endometrium
  - Eventually, sudden withdrawal bleeding occurs, which is painless, prolonged, excessive after 6-8 weeks of amenorrhea
- **Pathology:**
  Gross: symmetrical uterine enlargement
  Microscopic: swiss cheese appearance and hyperplastic myometrium
- **C/P:**
  Symptoms: painless prolonged excessive bleeding after amenorrhea for 6-8 weeks
  Signs: symmetrical enlargement of uterus , cyst in ovary
- **Investigations:**
  US show thickened endometrium and ovarian cyst
- **DD:**
  Complications of pregnancy
- **Treatment:**
  1. General: hemostatics, iron, NSAID
  2. Hormonal: gestagens (21 days not 10) as medical curettage for 3-6 months, NO OCPs
  3. Surgical : D&C, hysterectomy (in perimenopausal) , ablation (patients unfit for surgery)

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7. Diagnosis and management of DUB.

- Diagnosis = C/P & investigations.
- Diagnosis of DUB is a retrograde one, established only after exclusion of all structural and non- structural causes of AUB **(PALM COEIN).**
- This only could be achieved by:

1. *Careful history for:*
   Exclusion of pregnancy and its complications, medical disorders, endocrine disorders, systemic disease and iatrogenic causes as use of OCPs, IUD and anticoagulant therapy.
2. *Clinical Examination:*
   General, abdomen, vaginal, speculum for exclusion of uterine and adnexal masses and non-uterine causes of bleeding as rectal, urethral, vulvar and vaginal aetiology
3. *Laboratory tests and Hormonal assays:*
   For exclusion of liver disease, endocrine disorders and blood dyscrasias (as ITP and von Willebrand disease)
4. *Exclusion of malignant uterine and cervical neoplasms (Biopsy).*

The gold standard in diagnosis DUB:

1. Pelvic US: To exclude uterine and adnexal pathology.
2. Office hysteroscopy: To exclude small EP.
3. Endometrial biopsy: To diagnose EH.

Treatment of DUB:

It will depend on the underlying cause, the age of the patient, desire for fertility, the severity of bleeding and the acute and chronic nature of bleeding.

1. *During an acute attack:*

   Control bleeding by:

   - Antifibrinolytics: as Tranexamic acid, 500 mg t.d.s orally
   - NSAIDs: as Mefenamic acid, 500 mg t.d.s orally
   - D & C operation: To stop bleeding and obtain endometrial biopsy to exclude EH
2. *Hormonal ttt:*

   To induce regular cycles of average amount of bleeding:

   - Cyclic combined EE\PRG: e.g. OCPs for 21\month
   - Cyclic gestagens: oral tablets 10 mg daily\ for 10-14 days\month
   - LNG – IUD: effective method for inducing hypomenorrhea in these cases
3. *Surgical ttt:*
   - D & C: for control of acute attack, and obtaining biopsy to exclude EH\EC
   - Endometrial ablation: for recurrent DUB in absence of EH
   - Hysterectomy: Abdominal, vaginal, or laparoscope for menopausal causes of DUB with failed medical and\or HT, associated uterine or adnexal pathology, POP, or when D&C biopsy reveals a premalignant lesion as complex and atypical EH.

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8. Surgical ttt of DUB.

See previous question + details of endometrial ablation

Endometrial ablation:

- Is a procedure that aims at destroying the majority of endometrium, thus controlling blood loss.
- It carries an overall 90% success rates in reduction of uterine blood flow, and may induce amenorrhea in many cases.
- Ablation can be performed via hysteroscopic resection, hydrothermal balloons, or laser ablation.
- Endometrial ablation may be appropriate for persist or recurrent DUB in premenopausal women, with failed HT, in absence of EH or malignancy
- Cases with recurrent or irregular bleeding after ablation are best managed by hysterectomy.

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9. Enumerate 5 causes of menorrhagia.

1. Leiomyomas (SMM & ISMC large)
2. Adenomyosis
3. Endometrial polyps
4. Endometrial hyperplasia
5. IUD induced bleeding & DUB

NB

Scheme for any type of bleeding

i. *According to the cause:*

1. **Organic:**
    a. **General:**
        - HTN
        - Purpura
        - Congenital
        - Drugs
        - Renal failure
        - Liver failure
        - Anemia
    b. **Local:**
    - ***Obs:***
        - Early pregnancy
        - Antepartum hge
        - Postpartum hge
    - ***Gyna:***
        - Congenital: septate, bicornuate uterus
        - Traumatic: IUD, pessary
        - Inflamatory: acute, chronic
        - Neoplastic: benign, malignant & premalignant
        - Others: endometriosis, adenomyosis, displacements (RVF, prolapse, inversion), iatrogenic (OCPs)
2. **Functional (DUB):**
    a. Ovulatory (cyclic)
    b. Anovulatory (acyclic)

ii. *According to age:*

1. **Childbearing period:**
    a. Pregnancy complications
    b. Complications of contraception
    c. Local
    d. Dysfunctional
2. **Perimenopausal:**
    a. Commonest is DUB
    b. Most serious is endometrial carcinoma
3. **Postmenopausal:**
    a. Commonest is atrophic endometritis
    b. Most serious is endometrial carcinoma
    c. HRT
    d. Ring pessary for prolapse
    e. General: as urinary, rectal, …

iii. *<u>According to pattern:</u>*

	Local	**Systemic**	**Functional**
Menorrhagia	Congenital in uterus	Anything	Ovular
Polymenorrhea	Congenital in ovaries	-	Anovular
Metrorrhagia	Tumors + ulcers	Drugs	Anovular
Contact bleeding	Infection + tumors	-	-

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10. Causes of postmenopausal bleeding.

1. Iatrogenic: Hormonal therapy
2. Benign conditions: Atrophic vaginitis, atrophic endometritis, vulvar dystrophies, endometrial and endocervical polyps
3. Premalignant lesions: EH and high grade CIN lesions
4. Gynecologic malignancies: EC, cervical cancer, estrogen producing ovarian tumors, vaginal and vulvar cancers.

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11. Give an account on causes, investigations & treatment of contact bleeding.

Definition:

Bleeding that occurs on touching the crvix , ususlly postcoital following PV or cervical examination .

Common causes of CB:

"Any lesion in cervix or vagina"

1. *Cervix:*
   Itis, CIN, polyp
2. *Vagina:*
   Itis, cancer, VAIN
3. *Others:*
   Uterine polyp, granulation tissue after hysterectomy

Diagnosis:

General workup of AUB, but give special care to:

1. Speculum examination: to exclude cervical & endometrial polyps & erosion.
2. Pap smear test: for diagnosis of high grade CIN lesions.
3. Colposcopy: whenever a pap smear is suspicious, & biopsy from abnormal lesions.

Treatment:

According to lesions:

1. Cauterization for cervical erosions (ectopy).
2. Excision of cervical & endometrial polyps protruding from the cervix.
3. Management of high grade CIN lesions (ablation or LEEP excision).
4. Cervical & vaginal cancers: according to each type.

Anovulation and hirsutism

1. Detection of ovulation.

1. *<u>Ultrasound monitoring of ovulation (Folliculometry):</u>*

   Serial TVS (Transvaginal Sonogram) scans, in the follicular phase, allow monitoring growth, maturation and ovulation (rupture of dominant follicles at a mean diameter of 17-24 mm), and tracing early CL formation.

2. *Basal body temperature charts (BBT):*

   A temperature rise in temperature is expected in luteal phase because of the progesterone induced thermogenic effect. Although these charts are easily applicable, yet have the disadvantage of being easily manipulated by other factors that affect temperature.

   - **<u>Ovulatory cycles</u>**

     Show a biphasic BBT chart with a rise in temp. (0.2-0.3 C) in the luteal phase of the cycle. This temp. rise declines few days before the next cycle.

   - **<u>Anovulatory cycles</u>**

     Will show a flat monophasic BBT chart due to absence of thermogenic progesterone thermal effect.

3. *Mid luteal serum progesterone assay (MLSP): (measured by RIA "Radio imuuno-assay")*

   MLSP is performed 7 days after ovulation, or 21 days of the cycle (maximum CL (Corpus luteum) activity.

   - Levels <5 ng/ml suggest anovulation.
   - Values >10 ng/ml suggest ovulation with adequate CL function.
   - Levels 5-9 ng/ml suggest ovulation but with inadequate CL function (LPD) (Luteal phase deficiency).

4. *Urinary LH kits:*

   These can detect preovulatory LH surge in urine but cannot confirm rupture of the mature follicles and ovulation. They are primarily used in infertile couples to predict best timing for coital relation to improve the chances for oocyte fertilization

5. *Premenstrual endometrial biopsy (PEB):*

   PEB can be performed without cervical dilatation as an office procedure, using a small caliber cannula (Pipelle), or under general anaesthesia if cervical dilatation is mandatory.

   PEB done 2-3 days prior to menstruation will reveal either;

   - Secretory endometrium, in ovulatory cycles doe to progestational effect of the CL
   - Proliferative endometrium, in anovulatory cycles
   - Poor secretory changes, in cases of LPD

6. *Vaginal cytology and cervical mucous changes:*

   See before

7. *Laparoscopy:*

   Visualization of stigma of ovulation

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2. Define ovulation and drugs of its induction.

Definition:

Ovulation is the release of an egg, or ovum, which may then be fertilized by a sperm cell or dissolved during menstruation.

Drugs that induce its induction:

1. *Clomiphene Citrate (CC):*
   - A non steroidal compound closely related to diethyl stilnosterol (DES), but has no estrogenic effect on short term use.
   - **Mode of action:**
     - It competes with endogenous E2 for its hypothalamic receptors leading to their blockage. The hypothalamus reacts by increasing its pulsatile release of GnRH to combat this artificially induced hypo-estrogenic state.
     - Increased GnRH pulse frequency and amplitude increase pituitary FSH production, which directly stimulates follicular growth and maturation leading to production of increasing amounts of E2 levels.
     - Markedly elevated E2 levels will exert a positive feedback on Lh stimulating a strong LH surge (after CC has been stopped), finally leading the release of one or more of the mature oocytes (ovulation).
   - **Dosage:**
     50 mg oral tablets, twice daily for 5 days starting fifth day of menstruation\. Dose can be increased up to 200 mg/ day (4 tablets)
   - **Indications:**
     CC is the first line of treatment in anovulatory conditions with normal FSH production and intact hypothalamic pituitary axis as in cases of PCOD (Poly cyctic ovary disease), and post pill amenorrhea.
   - **Success rates:**
     CC is siccesful in induction of ovulation in around 85% of cases
   - **Side effects:**
     - Vasomotor flushes, nauseam headache and visual disturbances
     - Increased risk of twin pregnancy (10%) and multi fetal pregnancy (1%)
     - Ovarian hyperstimulation (OHSS): grade 1-2, with mild pelvic pain and discomfort
     - Anti estrogenic effects on the endometrium may cause hostile cervical mucous only repeated use

N.B.

- ***Tamoxifen:***
  - ***A weak anti estrogen (used in treatment of estrogen receptor positive breast cancers after mastectomy) that acts by mechanism similar to CC***
  - ***Dosage: 10-40 mg daily orally (1-4 tablets) or 5 days starting from the second day of cycle***
- ***Cyclofenil:***
  - ***A compound chemically related to CC with a weak estrogenic effect***
  - ***Dosage: 400 mg twice daily orally for 5 days starting 5th day of the cycle***
- ***Letrozole:***
  Used clinically

2. *Pituitary gonadotropins (FSH and LH derivatives):*
   A. *Human Menopausal Gonadotropins (HMG):*
   Derived from urine of menopausal women. It contains 75 IU FSH + 75 IU LH
   B. *Purified urinary FSH:*
   Derived from urine of menopausal women and contains only one IU of LH (75 IU FSH +

one IU LH). They are more suitable for cases with high endogenous LH as those of PCOD, to minimize possibility of severe OHSS (ovarian hyperstimulation), and in IVF (in vitro fertilization)/ICSI (intra cytoplasmic sperm injection) protocols.

C. *Synthetic FSH:*

FSH is prepared by recombinant DNA technology, used mostly in IVF/ICSI protocols in same indications as purified urinary FSH.

- **Mode of actions of GT:**
  Direct stimulation of growth and maturation of ovarian primordial folliclesm with production of increasing amounts of E2
- **Indications:**
  - CC resistant cases
  - cases with hypogonadotrophic anovulation (low FSH/LH levels)
  - In ICSI/IVF/ET protocols to stimulate growth of multiple follicles
  - Purified and synthetic urinary FSH are suitable both for cases with high endogenous LH (as PCOS), and in ICSI protocols to help retrieval of the largest number of oocytes.
- **Dosage:**
  Repeated I.M. Injections given from mid-follicular phase of the cycle until complete follicular maturation. Doses are repeated with TVS monitoring of follicular growth to avoid the complications of severe OHSS.
- **Side effects:**
  Same as those of CC, however;
  - OHSS is more frequent specially sever forms (grade 3-4 OHSS)
  - Marked increased risk of twin and multifoetal pregnancy (10-30%)
  - No adverse effects on cervical mucous or endometrium

3. *Human chorionic gonadotropins (hCG): (single I.M. injections)*
   - **Preparations and action:**
     hCG is prepared from urine of pregnant women. It has a strong LH action that induces an artificial LH surge leading to ovulation and oocyte release.
   - **Indication:**
     To assist ovulation specially in CC or HMG induced cycles
   - **Dosage and timing:**
     2 ampoules 5000 m/IU each, given by I.M. injection as one shot after full oocyte maturation (size of dominant follicle > 18 mm by TVS)
   - **Side effects:**
     OHSS, especially if given after HMG (Human menopausal Gonadotropin)
4. *Gonadotropin releasing hormone (GNRH) analogues:*
   - **Preparations:**
     GnRH agonists are synthetic decapeptide forms of GnRH, given either by repeated S.C. injection or as nasal spray.
   - **Dosage & mode of action:**
     In small repeated doses they act as GnRH thus increasing FSH production and inducing follicular maturation. In larger doses (long acting preparations), they induce initial FSH stimulation, followed by down regulation of hypothalamic receptors, with consequent suppression of FSH & LH production.
   - **Indications:**
     They are used mostly in IVF & ICSI protocols to prevent premature ovulation via LH

suppression, allowing for timed retrieval of maximum number of eggs.

5. *Combined therapy:*
   A. *CC/HMG/hCG:*
      Adding CC to HMG/hCG has the benefit of minimizing the dose of HMG thus reducing both the cost and chances of OHSS
   B. *GnRH/HMG/hCG :*
      Used in practice in various doses and different protocolsof stimulation special in IVF/ICSI stimulation protocols
6. *Adjuvant drugs used to assist in induction of ovulation:*
   A. *Bromocryptine:*
      0.2 mg 1-2 tablets daily, or Cabergoline: 0.5 mg ½ tablet twice weekly in cases of hyperprolactinaemia
   B. *Metformin:*
      500-800 mg daily in cases of insulin resistance
   C. *Thyroid extract:*
      Eltroxin 50-150 microgram daily in cases of hypothyroidism
   D. *Corticosteroids:*
      In cases of Addison's disease, adrenogenital syndrome, and PCOS

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3. Treatment of anovulation.

1. *Medical treatment for induction of ovulation:*
   See before.
2. *Surgical treatment for induction of ovulation:*
   Laparoscopic Ovarian drilling (LOD):
   - A surgical procedure in which a diathermy needle is used to make multiple small punctures in the surface of the ovary via laparoscopy
   - **Effect of drilling:**
     May decreased ovarian androgen environment resulting in a spontaneous ovulation, or at least minimize doses of CC/HMG required for stimulation
   - **Indications:**
     LOD is reserved to selected PCOs cases that are resistant to stimulation or ay require large doses of HMG with high costs and increased risk of OHSS
   - **Disadvantages:**
     Time limited effect on ovulation (3-6 months), and its potential for creating ovarian and peritubal adhesions

---

4. Ovarian hyperstimulation syndrome (OHSS): definition, grades, prevention and treatment.

Definition:

An iatrogenic disorder that describes the occurrence of ovarian multicyctic enlargement secoundary to the use of drugs for induction of ovulation, namely HMG/hCG.

Grades:

It is classified into 4 grades according to severity of symptoms:

i. *Mild forms (grades 1-2)*
   - These cases may be associated with variable degrees of abdominal pain and distension,

edema, mild ascites, hypotension, and oliguria

- Cases are managed by rest, reassurance, follow up, and symptomatic treatment

ii. Severe forms (grades 3-4)

- These cases may be associated with fluid shift from intravascular to extravascular compartment leading to severe ascites, pleural effusion, hypoalbuminemia, electrolyte imbalance, haemoconcentration, and changes in blood coagulability with tendency to DIC and increased risk of pulmonary embolism
- These life threatening cases are best managed by hospitalization, restoration of fluid and electrolyte imbalance, use of anticoagulants and sometimes tapping for aspiration of ascites. Diuretics are avoided as they wil increase haemoconcentration with risk of thrombosis
- Prevention of severe OHSS could be achieved by both careful TVS monitoring of HMG doses during stimulation, and withholding hCG injections in cases with Grade 2-3 hyperstimulation.

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5. What are the common clinical and hormonal findings in PCOS.
Diagnosis and long term risks of PCOS.

Diagnosis of PCOS:

1. *Symptoms:*
   Amenorrhea, DUB, infertility
2. *Signs:*
   a. **General:**
      Obesity, hirsutism and acanthosis negricans (insulin insensitivity)
   b. **Abdominal:**
      Not important
   c. **Local:**
      Associated follicular cysts
3. *Investigations:*
   a. US:
      - The ovaries are increased in size and volume
      - The ovaries show central dense stroma surrounded by small follicles (2-10 mm in diameter) peripherally arranged giving the characteristic **necklace appearance**.
      - No dominant or mature follicles are present due to chronic anovulation
      - US (Ultrasound) picture of PCOS may be encountered in up to 25% otherwise normal females

   b. Laboratory:
      Hormones, fasting insulin, glucose (fasting and postprandial), lipid profile
   c. Invasive:
      Laparoscopy and Endometrial biopsy in long standing cases (EH and EC)

Long term risks of PCOS:

1. Increased risk of diabetes mellitus, obesity, hyperlipidaemia, and cardiovascular disease
2. Increased risk of endometrial hyperplasia (EH); due to increased estrogen effect on the endometrium unopposed by progesterone
3. Increased risk of endometrial carcinoma if atypical Eh develops

6. Define PCOS, pathogenesis, and treatment

Definition:

The more recent description of Stein-Leventhal syndrome which is characterized by **2 or more of:**

1. Chronic anovulation: presenting clinically by 2ry amenorrhea or oligomenorrhea
2. Hyperandrogenism: (hirsutism, elevated serum LH, and increased free testosterone levels)
3. Characteristic ultrasound morphology: increased ovarian size and volume, with peripherally arranged small follicles in dense stroma (necklace appearance)

Pathogenesis:

A triad of:

1. *High LH:*
   - **Aetiology:**
     It is due to increased LH pulse and frequency. It is manifested by an abnormally high LH/FSH ratio (>2:1)
   - **Effect:**
     It stimulates secretion of androgen by ovarian theca cells, and inhibits aromatase enzyme responsible for conversion of ovarian androgen into estrogen, resulting in increased ovarian androgen.
2. *Hyperandrogenaemia:*
   - **Aetiology:**
     It is due to stimulation of theca cells (by both high LH and high insuin levels), together with inhibition of aromatase enzyme
   - **Effect:**
     Excess ovarian androgen leads to:
     a. Atresia of developing follicles leading anovulation and amenorrhea
     b. Hyperandrogenaemia; high serum androgen
     c. Hirsutism
     d. Excess androgen is converted in fat cells into estrone (peripheral conversion). This results in acyclic increase in estrone which leads to
        - Increased pituitary LH, and suppression of FSH
        - Unopposed estrogen leads to endometrial hyperplasia or carcinoma
3. *Hyperinsulinaemia:*
   - **Aetiology:**
     PCOS may be associated with peripheral insulin resistance disorder leading to hyperinsulinaemia
   - **Effect:**
     a. Increased sensitivity of ovarian theca cells to LH, thus increasing LH induced androgen production by the ovaries
     b. Decreased aromatase enzyme activity leading to excess ovarian androgen production
     c. Decreased production of SHBG(sex hormone binding globulin) leading to increased free androgen substrate

Treatment:

1. *Weight reduction:*

In obese females a reduction of 5-10% of body weight, reduces insulin and androgen and improves response to therapy, and may by itself re-establish ovulation

2. *Hormone therapy:*

   <u>In cases with menstrual disorders or DUB;</u>

   a. **<u>Cyclic gestation therapy</u>** for 10 days every cycle (day 16-25), to induce regular 28-30 days cycle (ex: medroxy progesterone acetate 10 mg per day)
   b. **<u>Combined OCP</u>** (day 5-25), to establish regular cycles in cases not requesting pregnancy

3. *Induction of ovulation for infertility:*

   Small notes about the titles of question no. 2

4. *Insulin sensitizing drugs: (metformin 500 mg/day orally)*

   Improve insulin sensitivity, thus decreasing hyperinsulinaemia and androgen levels. Such drugs also increase sensitivity of PCO to endogenous FSH and drugs like CC, and may by itself establish ovulatory cycles

5. *Corticosteroid therapy:*

   To suppress ACTH production in case of adrenal hyperandrogenism

6. *Surgical treatment via laparoscopic ovarian drilling (LOD):*

   Aims at decreasing ovarian androgen production. However, adhesion formation is an unfavorable side effect of surgery

7. *Hirsutism:*

   Cryproterone acetate, laser depilation, or electrolysis

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7. PCOS; pathogenesis, diagnosis, and treatment.
Discuss clinical presentation and diagnosis of PCOS.
Clinical picture and investigation of PCOS.
See before.

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8. Luteal phase defect: definition, aetiology, and treatment.

Luteal phase defect (1-2-3-3-4)

Definition:

A condition in which the endometrium shows poor secretory changes the luteal phase of ovulatory cycles. These cycles are characterized by either low progesterone levels in the luteal phase or short luteal phase duration (<11 days interval between ovulation and menstruation), or both.

Symptoms: (1)

Premenstrual spotting

Complications: (2)

1. Recurrent pregnancy loss
2. Infertility

Aetiology: (3)

1. Clomiphene citrate prolonged use for induction of ovulation
2. Hyperprolactinemia
3. Inadequate FSH/LH ratio at time of ovulation

Investigations: (3)

1. BBT
2. PRG mid-luteal serum level
3. PEB: thickness 5-9 mm

Treatment (4):

1. Progesterone support
2. HCG
3. Induction of ovulation
4. Anti- prolactin (e.g. bromocriptine, cabergoline)

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9. Etiology of hirsutism

1. *Idiopathic: increased hair follicles sensitivity to normal female androgen levels*
   - Increased receptor activity in the skin or
   - Increased activity of the enzyme 5 alpha reductase (responsible for the conversion of T(testosterone) into DHT(Di hydro testosterone) which has a more potent action)
2. *Adrenal gland causes:*
   - Congenital adrenal hyperplasia(partial or complete hydroxylase deficiency)
   - Adrenal tumors: secrete DHA (dehydroepiandrosterone), DHAS (dehydroepiandrosteronesulphate), and rarely Testosterone
3. *Ovarian causes:*
   - PCOS, hyperthecosis, and stromal cell hyperplasia (increased ovarian androgens.)
   - Androgenic ovarian tumors as sertolileydig cell tumor, adrenal rest tumore, hilar cell tumore, and gonadoblastoma. (increased testosterone production)
4. *Mixed ovarian and adrenal hyper androgenism: (30-40% of cases)*

   Increased adrenal production of androgen leads to inhibition of follicular maturation & induction of premature atresia with increased production of ovarian androgen
5. *Pituitary gland:*
   - Cushing's syndrome due to increased production of ACTH
   - Acromegaly due to increased production of GH
6. *Androgenic drugs:*

   Rarely, long term use of drugs with androgenic side effects (as Danazol in endometriosis) may cause hirsutism and virilizing effects

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10. Treatment of hirsutism.

1. *Elimination of specific causes:*
   - Removal of androgen secreting ovarian or adrenal tumors
   - Elimination of drugs suspected to the contribute to the abnormal hair growth
   - Treatment of cushing's syndrome, thyroid disease, or hyperprolactinaemia
2. *Hair removal techniques:*
   - Shaving, tweezing, waxing, and use of depilatories, performed at repeated intervals
   - Bleaching is effective for mild hair growth
   - Permanent destruction of hair follicles by electrolysis or by laser is reasonably effective
3. *Suppression of androgen synthesis:*
   - **<u>Oral contraceptive pills (OCPs):</u>**
     Containing combined low does E and P, decrease ovarian androgen production, increases

SHBG, and decrease free T levels. Progestins may also nhibit 5-alpha reductase activity

- **Corticosteroids (dexamethasone 1-5 mg/day):**
  Induce suppression of adrenal androgen production in severe cases of CAH. Long term side effects include osteoporosis, diabetes mellitus, and avascular necrosis of the hip
- **Spironolactone:**
  An aldosterone antagonist used frequently as diuretic that also inhibits 5-alpha reductase and variably suppresses ovarian and adrenal synthesis of androgen
- **Cypretorone acetate:**
  A potent progestin and antiandrogen that inhibits LH and decreases androgen levels. It is used for 10 days each cycle. Diane 35 is an OCP that uses cypretorone acetate ass a progestin and is widely used in treatment of hirsutism in females that request contraception

4. *Androgen receptor blocker*
   - It inhibit binding of DHT to androgen receptors thus directly inhibiting hair growth. When combined with OCPs or progestins further benefit may be obtained.
   - Cimetidine: competes with androgen at the receptor site. Dose: 300 mg 5 times daily

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11. Hirsutism: etiology, investigations, and treatment.

Etiology & treatment:

See before.

Investigations of a case of hirsutism:

1. *Hormonal assay:*
   - Plasma T level (n=0.2-0.8ng/ml), levels >2ng/ml suggest androgen secreting tumor.
   - Free T level (n= 1-3% of total T) it is a good index of androgenicity
   - DHAS (dehydro-epiandrosterone sulphate) (n=1500-2500 ng/ml), levels >9000 ng/ml suggests adrenal tumor
2. *Radiological assay:*
   - CT or MRI on the pituitary gland
   - IVP and abdominal US for adrenal tumor
   - Pelvis US, for PCOS, and virulizing ovarian tumors

Infertility

1. Discuss cervical factors of infertility (2010).

Causes:

They may impair sperm transport through cervical canal making it incapable of fertilization:

- Changes in physical & chemical properties e.g.: ↓amount or ↑ viscosity.
- Infection & pus cells e.g.: acute or chronic cervicitis.
- Anti-sperm antibodies in cervical mucus.
- Destruction of mucus secreting glands b conization or excessive cauterization.
- Elongation of cervical canal by a cervical fibroid (rare).

Assessment:

1. **Assessment of physical properties of cervical mucus**

Pre-ovulatory	Post-ovulatory
Thin, profuse, watery Acellular, Alkaline	Thick, scanty Cellular
+ve thread test (stretch 6-10 cm) Max. fering when dried under microscopy	-ve thread test (threads break < 6 cm) Min. fering when dried under microscopy

2. **Post.coital test (PCT):**
   - Examines cervical mucus 6-10 h after intercourse (at time of ovulation).
   - Assesses number of living & dead sperms and presence of leucocytes (normally > 20 progressively motile sperm /HPF)
   - Abnormal PCT may be dt: Hostile cervical mucus- Anti sperm ant-bodies
   - Limited prognostic value.

Management:

- Treatment of the cause as chronic cervicitis, hostile cervical mucus, polyp removal.
- Estrogen & mucolytics: improves cervical mucus physical properties.
- IUI: bypassing the cervix through injection of processed semen directly within the endometrial cavity.

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2. Discuss uterine causes of infertility.

10-15% of female causes via these mechanisms:

1. Interference with sperms transport through endometrial cavity.
2. Bilateral cornu obstruction of tubal ostea.
3. Impairment of blastocyst implantation within endometrial cavity.

1. *Congenital:*
   - Septate and bicornuate uterus (infertility &/ or RPL)
   - Uterine hypoplasia or aplasia (amenorrhea & infertility)
2. *Intrauterine synechae dt:*
   - Over curettage of basal endometrial layer (Ashermann's syndrome).
   - Acute septic endometritis (puerperal, post-abortive sepsis, IUD associated).
   - Chronic specific infections e.g.: TB endometritis.
3. *Uterine leiomyomas* especially large and multiple ISM and SMM.

4. *Uterine polyp:* SMF polyps or endometrial polyps.

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3. Investigations of infertile couples.

i. For male infertility:

Male should be evaluated first before going into invasive female investigations.

1. Semen analysis:
   - Should be obtained from all males.
   - Collected by masturbation after 3-4 days of abstinence from intercourse.
2. Hormonal assay: FSH, LH, prolactin, testosterone.
3. Doppler US: on testicles to detect varicocele.
4. Testicular biopsy: differentiate between defective spermatogenesis and obstructive disorders.
5. Chromosomal studies: diagnose genetic disorders.

N.B:

Criteria for normal semen analysis:

- Volume: 2 ml or more
- Count: 15 million/ml
- PH: 7.2-7.8
- Motility: 50% or more with forward progressive motility.
- Morphology: 30 % or more with normal morphology.

ii. For female infertility:

1. For ovulation detection:

See before (anovulation).

2. For uterine factors:

a. Pelvic US: (the gold standard) (TAS-TVS-3DUS-SIS)

Diagnosis of:

- Congenital uterine anomaly: septate, bicornuate uterus (TVS-3DUS).
- Uterine leiomyoma (TAS-TVS-3DUS).
- SMM, SMF polyp, endometrial polyp (3DUS, SIS)
- Intrauterine adhesions (SIS).
- Associated adnexal mass (TAS, TVS).

b. Hysterosalpingography (HSG):

- Entails injection of radio-opaque dye → visualize endometrial cavity & fallopian tubes.
- Gold standard in diagnosis of:
  - Mullerian anomalies e.g.: septate, bicornuate, unicornuate uterus.
  - Endometrial pathology e.g.: polyp, SMM, intra-uterine adhesions.
  - Tubal obstruction.
  - Peritubal & peritoneal adhesions.

c. Hysterscopy:

Allows direct visualisation of uterine cavity and minor surgical procedures.

d. Premenstrual endometrial biopsy (PEB):

To exclude TB endometriosis in suspicious cases.

3. **<u>For tubal & peritoneal.</u>**
    a. Hystersalpingography: diagnose:
        - Unilateral or bilateral, distal or proximal tubal obstruction.
        - Hydrosalpinx: distal tubal obstruction with dye filled sacculation.
        - Suspected pelvic adhesions; loculation of the dye in the pelvis in control film.
    b. Laparoscopy & dye injection:
        Advantages:
        - Accurate diagnosis of fimbrial, peritubal, pelvic adhesions.
        - Diagnosis of pelvic pathology as endometriosis, PID, TB peritonitis.
        - Diagnosis of ovarian pathology as PCO, endometrioma.
        - Operative laparoscopy can be used in:
            – Lysis of pelvic adhesions.
            – ttt of pelvic & ovarian endometriosis.
            – Ovarian drilling in PCO.
4. **<u>For cervical factors: see Q 1</u>**
5. **<u>Hormonal assay</u>**
    a. Serum FSH & LH (day 1-3): abnormal LH/FSH ratio in PCO.
    b. Urinary LH: +ve result denotes impending ovulation (24 h).
    c. Mid-luteal serum PRG
    d. Serum prolactin: normally 2.8-28 ng/ml), high levels denotes hyperprolactinemia.
    e. Serum androgen (total & free T, DHEA) in cases of hirsutism & PCO.
    f. Thyroid function tests (TSH, T3, T4): to detect major thyroid disorders.

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4. List the indications of IVF/ICSI procedures.

1. Severe tubo-peritoneal damage and\or extensive tubo-peritoneal adhesions.
2. Failed adhesiolysis and\or tuboplastic surgery.
3. Severe male factor as severe oligospermia and azospermia (due to obstruction).
4. Unexplained infertility especially after repeated failed intra uterine insemination (IUI).

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5. Male factor of infertility.

The male is responsible alone for up to 40% of infertility cases, and should be evaluated separately and thoroughly in every couple presenting with infertility, before going into invasive female investigations.

Aetiology:

A) Abnormal spermatogenesis:

<u>1. Increased scrotal temperature:</u> (Scrotal temperature should be 1°C less than body temperature)

- Undescended testes

- Varicocele

<u>2. Genetic causes:</u>

As in micro-deletion of the Y chromosome

<u>3. Drug induced:</u>

As in anti-hypertensive drugs, anti-epileptics, psychotropic agents, prolonged antibiotic administration and chemotherapeutic agents.

B) Failure of sperm transport through the vas deferns:

1. Bilateral epididymeal obstruction:

Gonorrheal inflammation is the commonest cause.

2. Bilateral vas ligation:

- Intended: as in male sterilization
- Iatrogenic: rarely faulty ligation during bilateral inguinal hernioplasty operations

3. Immotile cilia syndrome:

All sperms are immotile

4. Congenital absence of ejaculatory duct or spermatic cord

C) Failure of semen deposition (due to ejaculatory dysfunction):

- Impotence
- Premature ejaculation
- Retrograde ejaculation (in DM or disc prolapse due to autonomic nerves affection) and an-ejaculation

Diagnosis of male factor:

A) History:

- Related operative procedures: repair of inguinal hernia or varicocele.
- Infection by STDs, as gonorrhea, chlamydia,etc.
- Coital problems, as erection and ejaculation disorders
- Heavy smoking, due to decreased oxygen tissue perfusion
- Chronic drug intake as anti- hypertensives, anti-epileptics, psychotropic drugs, etc..

B) Clinical examination:

- To exclude: undescended testes, testicular masses and small testes.
- To detect: the presence of hydrocele, and/or varicocele and their extent

C) Special investigations:

1. Semen analysis: should be obtained from all males during infertility investigations.

"Samples are collected by masturbation after 3-4 days of abstinence from intercourse"

2. Hormonal assay: FSH, LH, prolactin and testosterone

3. Doppler US on the testicles to detect varicocele

4. Testicular biopsy: to differentiate between defective spermatogenesis and obstructive disorders

5. Chromosomal studies: to diagnose genetic disorders

Criteria for normal semen analysis (WHO 2010)

Parameter	Normal criteria
Volume	2 ml or more
Count	15 million/ml
Ph	7.2-7.8
Motility	50% or more with forward progressive motility
Morphology	30% or more with normal morphology

Term	Definition
Aspermia	Absence of semen
Azoospermia	Zero sperm count
Oligospermia	Count <15 million/ml
Asthenospermia	< 50% with forward progressive motility
Teratospermia	> 70% abnormal forms

Management of male infertility:

Improvement in poor semen parameters may take from 3-6 months of appropriate therapy

A. Stop smoking (to improve oxygenation)
B. Use of Multivitamins and Antioxidants, are thought to improve semen count and motility.
C. Treat erection disorders using medications (as Viagra) or synthetic prosthesis.
D. Shift to other types of anti-hypertensive and psychotropic drugs if they were the cause.
E. Hormonal treatment: CC & HMG are useful in cases of defective spermatogenesis.
F. Surgical treatment:
   - Ligation of varicocele: may improve sperm count and motility in some cases.
   - Artificial prosthesis: in some erection disorders.
G. Assisted reproductive techniques (ART):
   a. Intrauterine insemination (IUI) indicated in cases with:
      - Mild oligospermia
      - Coital dysfunction and erection disorders
      - Unexplained infertility
   b. In-vitro fertilization and embryo transfer (IVF/ET): suitable for:
      - Mild male factor
      - Unexplained infertility
   c. Intra-cytoplasmic sperm injection (ICSI/ET): suitable for severe male factor as:
      - Severe oligoasthenospermia
      - Obstructive azoospermia, sperms recovered directly by testicular sperm aspiration

   (Cases with genetic disorders & obstructive azoospermia carry the poorest prognosis)

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6. Unexplained infertility: etiology and management.

Unexplained infertility is a diagnosis of exclusion. It may be present in almost 15% of infertile couples when all standard male and female infertility investigations fail to detect a possible cause for infertility. Almost 60% of these couples will conceive within 3 years without treatment.

Possible contributing factors:

1. Immunological and psychological factors
2. Defective sperm fertilization capacity
3. Decreased ovarian reserve: decreased number and quality of oocytes (day FSH level > 10 mIU/ml, and low AMH < 1 ng/ml)
4. Occult cervical infection.

3. CONTRACEPTION

1. Barrier methods of contraception.

1. *Male and female latex condom:*
   - Latex condoms, used by the male, are the most widely used barrier contraceptive method all over the world. The female latex condoms are not widely available in many countries.
   - **Efficacy:**
     High pregnancy prevention rate up to 97% which is additionally increased when used with spermicidal creams, coitus interruptus or the safe period.
   - **Advantages:**
     High efficacy, ready availability, easy use, and cheap price
   - **Medical benefits:**
     Prevention of STDs (as HIV and HBV), HCV, and treatment of premature ejaculation
   - **Disadvantages:**
     Rarely becomes torn, or slips out, thus losing protection
2. *Spermicides (NONOXYNOL-9):*
   - Spermicides inactivate sperms deposited in the vagina during intercourse.
   - **Forms:**
     Vaginal tablets, creams, gel, or sponge applied 30 minutes before intercourse
   - **Efficacy:**
     Failure rates reach up to 30 /HWY if used alone however they are more effective when used with condoms or vaginal diaphragms
   - **Disadvantages:**
     May cause allergic vaginitis in some women

Common advantages of barrier methods:

1. Easy to initiate and discontinue.
2. No systemic side effects.
3. No effects on future fertility.

Other barrier contraception methods include: cervical cap& vaginal diaphragm.

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2. Complications of IUD.

3. Problems and complications associated with intrauterine contraceptive device use.

1. *Abnormal uterine bleeding*
   - *A. Post insertion bleeding*

     Mild bleeding or spotting may occur in the few days in infection. Bleeding usually stops spontaneously, but if persists use anti fibrinolytics and venotonics.
   - *B. Menorrhagia*
     - Heavy menstrual bleeding may occur in the first few cycles following IUD insertion, but may continue for a longer period in some cases.
     - **Etiology:**
       - Mostly due to increase PGLs production, or increased fibrinolytic activity.
       - Copper IUD increases blood loss by 35% in many women.
     - **Management:**
       - Exclude uterine and pelvic pathology: by clinical examination and pelvis US

- Anti fibrinolytic agents(Tranexamic acid 500 mg orally 2-3 times daily)
- And/or NSAIDs (Brufen 600 mg orally 2-3 times daily) till bleeding stops
- Remove IUD: if displaced or discovered significant uterine or pelvis pathology
- LNG-IUS induces endometrial atrophic changes, leading to 70% reduction in menstrual blood loss, and hence can be used as an alternative in cases who suffered intractable bleeding with the use of regular Cu IUD

C. Metrorrhagia

- Irregular inter menstrual bleeding is usually associated with anovulatory dysfunction, pelvic infection or partial expulsion of an IUD.
- **Management:**
  - Proper replacement of a cervically displaced IUD
  - Anti fibrinolytics and NSAIDs
  - Cyclic Gestagens (nor-ethisterone acetate 5 mg orally 2-3 times daily for day 10-15th every cycle for 2-3 cycles)
  - Combined estrogen/PRG therapy (OCPs 21 days each cycle for 2-3 cycles.

2. *Pelvic pain:*
   - **During insertion:** cramping pain is common due to forcible insertion. Dive NSAIDs
   - **After insertion:** possibly due to incorrect placement. Check position and replace properly
   - **Backache:** due to pelvic congestion, or chronic cervicitis
   - **Acute abdominal pain:**
     - With IUD is in situ; as with abortion, tubal ectopic pregnancy and PID
     - With a missed IUD; as in cases of uterine perforation
3. *Pelvic infection:*
   - Chronic cervicitis and cervical erosions are more common in presence of IUD.

     Management:
     - Antibiotics should be given then IUD removed
     - Cauterization of cervical erosion
   - PID is more common with IUDs ; the threads of an IUD may facilitate ascending infection, especially with STDs, causing endometritis and salpingitis.

     Management:

     Antibiotics then removal.
4. *Vaginal discharge:*
   - Increased watery vaginal discharge, due to pelvic congestion
   - Muco purulent discharge may occur in chronic cervicitis

     Management: treat the cause.
5. *Expulsion of an IUD:*
   - Expulsion may occur usually in the first few cycles after insertion of IUD due to:
     - Intermittent uterine contractions especially during menstruation, or
     - Misplacement at the time of insertion
   - Management: in both cases IUD should be removed and correctly re-inserted
6. *Perforation:*
   - Mostly occurs at the time of insertion.

- **Symptoms:**
  Sharp stabbing or colicky pelvic pain, together with persistent vaginal bleeding
- **Signs:**
  The threads will not be felt by the patient nor be seen by speculum examination
- **Investigations:**
  TVS will fail to detect the IUD within the uterine cavity, while a plain X-ray would reveal its presence in the pelvic cavity.

7. *Pregnancy on IUD:*
   - Failure rates for IUDs are very low < 0.5/HWY.
   - Most pregnancies occur when the IUD is displaced downwards intra-cervically, leaving the rest of the endometrial cavity unprotected.
   - **Management:**
     - If threads are visible, IUD is removed.
     - If threads are not visible, IUD is left in place with risk of abortion of around 50% with higher incidence of septic abortion.
8. *Ectopic gestation:*
   - IUD prevents intra uterine but not extra uterine pregnancy to the same extent.
   - Incidence of ectopic gestation may be slightly increased especially in the presence of PID.
   - **Management:**
     Depends on the patient's general condition, size of gestational sac, the condition of the tube and desire for future fertility.

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4. Missed IUD: causes and management.

Causes:

1. (+) Indrawn threads.
2. Pregnancy.
3. Perforation.
4. Expulsion.

Management:

If IUD threads are not felt by PV nor seen at speculum examination, a TVS should be performed to confirm the presence or absence of the IUD within the uterine cavity, and exclude pregnancy.

1. If the IUD is intrauterine; leave it in place if there is no other indication for removal
2. If IUD is not detected by TVS; a plain X-RAY to the abdomen and pelvis is performed:
   a. Absence of IUD on plain x-ray denotes expulsion outside the body. A new IUD maybe inserted if the patient asks for one
   b. Presence of IUD on plain X-RAY denotes perforation and presence inside the abdominal or pelvic cavity. Such cases are managed by removal of the IUD by laparoscopy or laparotomy, as its presence will provoke an inflammatory reaction leading to peritoneal, omental, and intestinal adhesions, and/ or injury

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5. Contraindications of contraceptive pills.

Absolute contraindications	Relative contraindications
1. Thrombophlebitis or thromboembolic disease 2. History of DVT 3. Coronary heart disease 4. Cerebrovascular accidents or strokes 5. Liver disease (impaired function) 6. Malignancy of the female genital system 7. Abnormal bleeding from the genital tract 8. Benign or malignant liver disease 9. Suspected or known carcinoma of breast or history of benign neoplasms of the breast	1. Superficial thrombophlebitis 2. Varicose veins 3. Migraine headache 4. Hypertension 5. Diabetes mellitus 6. History of liver disease 7. Gall bladder stones 8. Age >35 years 9. History of: Pre-eclampsia, Diabetes with pregnancy, Cholestasis with pregnancy

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6. Mode of action of contraceptive pills.

This type of contraception contains a combination of both estrogen and gestagen in the form of oral pills, vaginal rings or adhesive dermal patches. They exert their contraceptive effect through the following mechanisms;

1. **Estrogen:** inhibits ovulation via suppression of GnRH, FSH, and LH surge
2. **Gestagen:** synthetic progesterone exert their effect through;
   - Endometrial changes unfavorable for implantation: due glandular atrophy and stromal edema
   - Cervical mucus changes resulting in thick cervical mucus hostile to sperm penetration
   - Altered tubal motility and secretions; unfavorable to oocyte transport
   - Interference with ovulation; through FSH& LH suppression (but less than estrogen)

---

7. Side effects of combined oral contraceptive pills.

1. *Spotting:*

   Mostly due to inappropriate hormone content of the pill. In such cases shift to other preparations of higher dose of gestagen in the following cycles.
2. *Breakthrough bleeding:*

   Inter-menstrual bleeding during the course of pill taking. If persists more than 3 cycles, then shift to higher hormone content pills.
3. *Hypo-menorrhea:*

   It is common with OCP as gestagens cause glandular atrophy. Pills should be stopped if the symptom is unacceptable by the user.
4. *Amenorrhea:*

   During intake or after stopping the pills, is managed by excluding pregnancy, inducing withdrawal bleeding by gestagens, and shifting to another non hormonal method.
5. *Thrombo-embolic disorders:*

   Increased due to increased platelet adhesiveness, increased level of factors II, VII, IX AND X.

The risk for DVT and pulmonary embolism is increased from 4 to 6 folds if OCP were not stopped at least 4 weeks before surgery.

6. *Hypertension (HTN):*

   Increased incidence in predisposed patients after prolonged use.

7. *Diabetes mellitus (DM):*

   Impaired carbohydrate metabolism may predispose to DM, and in diabetic patients will lead to difficult glycemic control

8. *Liver:*

   Transient impairment in liver functions is not uncommon, with increased hepatic cholestasis, higher incidence of gall stones, and rarely benign hepato-cellular adenoma.

9. *Effect on lactation:*

   Decreased milk production and quality protein only with COCs.

10. *Other minor side effects:*

- ***Nausea and vomiting:*** maybe encountered in the first few cycles
- ***Migraine headache:*** if severe, needs discontinuation and shift to non-hormonal method
- ***Irritability and depressive mood:*** shift to lower progesterone content pills
- ***Weight gain:*** usually minor, mainly due to salt and water retention
- ***Breast tenderness, and enlargement:*** shift to lower E/P pills
- ***Acne:*** may worsen during OCP intake
- ***Skin pigmentation:*** chloasma similar to pregnancy due to increased pigment deposition.
- ***Change in libido:*** decreased sexual desire may rarely occur in some women
- ***Vaginal discharge:*** leucorrhoea may occur due to associated cervical congestion
- ***Eye symptoms:*** corneal edema, contact lens wearers suffer from blurring of vision and corneal irritation. Reported cases of transient optic nerve ischemia, and transient blurring of vision. The previous condition call for the pills to be stopped.

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8. Advantages of Oral Contraceptive pills

i. *Contraceptive benefits of COCs:*

1. Most effective method of contraception 0.1-1/HWY (Hundred Women yearly). If pregnancy occurs it is said that it is rather patient failure than method failure
2. Excellent cycle control specially in patients with previous irregular cycles
3. No long term adverse effects on fertility, with rapid recovery after pill discontinuation
4. No effects on sexual intercourse unlike coitus interruptus, spermicidals, condoms, and calendar methods of the safe period

ii. *Non contraceptive benefits of COCs:*

1. Treatment of DUB (Dysfunctional bleeding)
2. Postponing next menstruation(delaying an expected cycle)
3. Treatment of spasmodic Dysmenorrhea
4. Less risk of PID (Pelvic Inflammatory Disease), functional cysts of the ovary, endometrial carcinoma, epithelial ovarian cancer, (both previous cancers have 50% decreased risk) and colorectal cancer

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9. Combined Oral Contraceptive pills advantages, side effects, risks and contraindications.

Advantages, side effects and contraindications: see before.

Disease risk associated with the use of COCs:

Disease	Relative risk
Ischemic Coronary Heart Disease(ICHD)	Slightly increased risk
Ischemic stroke	2 fold increased risk
Venous thrombo-embolism	3-5 fold increased according to type of gestagen
Breast cancer	Small increase in risk on prolonged use (time related)
Cervical cancer	Small increase in risk on prolonged use (in smokers)

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10. Progesterone only contraception: types and indications.

Types:

1. Progestogen only pills.
2. Progestogen only injectable contraception.
3. Sub-dermal progestagen implants.
4. Levo-norgestrel releasing intra uterine system (Mirena).

1. *Progestogen only pills:*
   - Each pill contains the same amount of gestagen (levo-norgestrel, nor-ethisterone, desogestrel), administered orally, daily; around the same time every day, without a pill free interval irrespective of menstrual cycle.
   - **Indications:**

     POPs are suitable for most women however they are most often used by women for whom COCs are contra-indicated (e.g; breast feeding, hypertension, age above 40 years, women who smoke regularly, and those who commonly get migraine headache especially with aura).
2. *Progestogen only injectable contraception:*
   - The most commonly used one is depot-medroxy progesterone acetate (DMPA).
   - **Indications:**

     Same as POPs especially if the patient is non-compliant can't take the pill daily around the same time.
3. *Sub dermal progestagen implants:*
   - A sub dermal implant is made from a non-biodegradable polymer which contains an active slow release progesterone formulation.
   - **Indications:**

     Same as POPs and injectables with the benefit of providing contraception for about 3 years.
4. *Levo-norgestrel releasing intra uterine system (Mirena):*
   - It's an IUD that releases 35 micrograms of levo-norgestrel daily.
   - **Indications:**

     Same as other types of progesterone contraception in addition, it's used for treatment of dysfunctional uterine bleeding.

11. Side effects and contra-indications of oral contraceptive pills.

See before.

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12. Contraception for lactating women.

1. *IUD:*

   An ideal method as it gives long term contraception Without interference With lactation.
2. *Progesterone only injectables:*

   DMPA: 150 mg IM/ 3 months are suitable in most cases.
3. *Progesterone only pills (POP):*

   Daily administered in the same time, without interval regardless presence or absence of menstruation, are good alternatives to injections.
4. *Barrier Method:*

   Male and female condom and diaphragm are suitable especially with back-up methods as spermicidals or coitus interruptus.
5. *Spermicidal gel or foam:*

   Used in cases with infrequent Intercourse (husband travelling …etc).
6. *Physiologic Methods:*

   Coitus interruptus is a popular method widely practiced.
7. *The safe period:*

   Not always suitable due to the high incidence of menstrual irregularity with lactation.

Other topics:

1. Physiological methods of contraception.

1. *Coitus interruptus*
   - It is Withdrawal and ejaculation outside the vagina during intercourse.
   - Efficacy of this method is not high; as in some cases the pre-ejaculatory fluid may contain sperms capable of fertilization.
   - It is best coupled with the safe period method.
2. *The safe period*
   - Intercourse is totally prevented, or protected (by a condom or coitus interruptus) at the time of expected ovulation (day10-18 of a cycle of 28 days), while allowed for the rest of the month without protection methods.
   - This method is suitable only for intellectual couples, with regular cycles.
   - Efficacy is good, and is improved when coupled with additional methods that allow for more accurate prediction of ovulation as the BBT charts and the urinary LH tests.
3. *Lactation*
   - During lactation, at least 60% of females will experience amenorrhea, anovulation, and oligo-ovulation during the first 6 months of the puerperium, due to elevated prolactin levels.
   - Efficacy: is not high (nearly 50% protection if used alone), due to the fact that some women may resume ovulation at variable unpredictable periods of time.
   - Efficacy is improved by adding other methods such as spermicidals, barrier, and coitus interruptus.

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2. Mechanism of action of IUD.

1. *Interference with Blastocyst implantation, rendering the endometrium unsuitable via:*
   a. Local foreign body inflammatory responses
   b. Increased local endometrial prostaglandin production
   c. Inhibition of carbonic anhydrase and alkaline phosphatase activity(copper IUD)
   d. Induction of atrophic endometrial changes(LNG-IUS)
2. *Interference with sperm motility rendering them incapable of fertilization through:.*
   a. Inhibition of sperm motility
   b. Inducing an anti-sperm phagocytic activity(Cu IUD)
   c. Toxic effects on sperms inhibiting its capability of fertilization(Cu IUD)
   d. Thick cervical mucus interfering with trans cervical sperm penetration.

---

3. Advantages of IUDs.

1. Single choice method, with long term protection (6-8 years).
2. It has no effect on future fertility.
3. It doesn't affect lactation.
4. It doesn't affect or interfere with sexual intercourse.
5. Very low failure rate: 0.5/HWY (hundred women yearly).

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4. Contra-indications of IUD.

1. Undiagnosed vaginal bleeding.
2. Uterine anomalies that interfere with proper insertion(bicornuate/ Septate uterus)
3. Uterine pathology that interferes with proper insertion or cause complications (leiomyoma/polyps/adenomyosis).
4. History of pelvic inflammatory disease or acute PID.
5. History of ectopic pregnancy.
6. Wilson's disease (copper IUD is contra indicated in this condition).

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5. Mechanism of action and side effects of different types of progesterone only contraception.

1. *Progestogen only pills:*
   - **Mode of action:**
     - They affect mainly cervical mucus rendering it thick preventing sperm penetration.
     - They induce atrophic changes of the endometrium.
     - They inhibit ovulation in 60% of cases.
   - **Side effects:**
     - Menstrual irregularities.
     - Ectopic pregnancy.
2. *Progestogen only injectables:*
   - **Mode of action:**

     They act mainly by inhibition of ovulation.
   - **Side effects:**
     - Vaginal spotting is common in the first few months.
     - Up to 70% will have amenorrhea by the end of the first year.
3. *Sub dermal progestagen implants:*
   - **Mode of action:**

     Mainly via inhibition of ovulation.
   - **Side effects:**
     - About 20% will have amenorrhea within 3 months.
     - Up to 50% will have frequent or prolonged cycles.
4. *Levo-norgestrel intra uterine releasing system(Mirena):*
   - **Mode of action:**
     - Interference with Blastocyst implantation, rendering the endometrium unsuitable via:

   a. Local foreign body inflammatory responses

   b. Increased local endometrial prostaglandin production

   c. Induction of atrophic endometrial changes

   "a & b are general mechanisms while c is specific for Mirena"
     - Interference with sperm motility rendering them incapable of fertilization through:

   a. Inhibition of sperm motility

   b. Thick cervical mucus interfering with trans-cervical sperm penetration.

- **Side effects:**
  Same complications of IUD minus the abnormal uterine bleeding

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6. Indications and side effects of female sterilization.

Indications:

1. Cases with medical or surgical diseases or conditions that contraindicate pregnancy and necessitate pregnancy termination if it occurs.
2. It should not be advised for cases that just need to limit their family number.

Side effects:

Pelvic congestion **(post ligation syndrome)** leading to heavy menstrual bleeding and pelvic pain.

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7. Emergency contraception.

Definition:

It is the use of methods to prevent pregnancy after an unprotected intercourse has occurred whenever no method has been previously used, or a method did not function properly such as a torn condom, or a missed pill.

Options for Emergency Contraception:

1. *IUD Insertion within 24-48 hours of unprotected intercourse.*
2. *OCPs: started immediately after intercourse 4 tablets (2 tablets /12 hrs):*
   A. *POP high dose pills regimen:*
      - Each oral tablet contains 0.75 mg of levo- norgestrel.
      - The first tablet should be taken within 72 hours after unprotected intercourse, and the 2nd dose tablet is taken 12 hours after the first one.
   B. *COCs regimen:*
      - Each dose should contain at least 0.1 mg of ethinyl estradiol+ 0.5 mg of levo-norgestrel i.e.; 4 tablets of the standard low-dose COCs taken together).
      - First dose must be taken within 72 hours after unprotected intercourse, with a repeat dose 12 hours after first dose.
      - Low-dose COCs = 4 pills per dose, High-dose COCs= 2 pills per dose

Side Effects:

Nausea, vomiting, headaches, dizziness, fatigue, breast tenderness, irregular bleeding and spotting.

Fibroid

1. **Types of bleeding with fibroid.**
   Incidence and causes of menorrhagia and causes of metrorrhagia in fibroid.

   A. *Menorrhagia:*
   - The commonest presentation (30% of cases).
   - Mostly with SMM and large multiple ISM.
   - Excessive bleeding maybe due to:
     a. Increased surface area of endometrium.
     b. Mechanical interference with uterine contraction.
     c. Associated endometrial hyperplasia (EH) or hormonal imbalance due to chronic anovulation.
     d. Increased myometrial vascularity (venous congestion and engorgement).

   B. *Metrorrhagia:*
   Maybe due to:
   a. Sloughing in the tip of SMF polyp.
   b. Associated disturbed pregnancy.
   c. Contact bleeding:
   A with PV, sexual intercourse).
   d. Postmenopausal bleeding:
   (Malignancy).
   e. Polymenorrhea
   (Due to ovarian congestion).

N.B. Amenorrhea with Fibroid = Pregnancy

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2. **Symptoms of uterine leiomyoma.**
   - ***A****symptomatic*
   - ***B****leeding*
   - ***C****omplications (infertility)*
   - ***D****ischarge (leucorrhea)*
   - ***E****nlargement*
   - ***3P*** *(**P**ain, **P**ressure & **P**regnancy complications)*

Age:
More prevalent between 35-45 years.
Parity:
More among infertile patients, nulliparous and low parity population.
No symptoms:
- The **majority of solitary and small fibroids are asymptomatic** (esp. SSM & ISM).
- The **nearer** the fibroid **to endometrial cavity**, the more the chances of being **symptomatic** even if relatively small (as small SMM and SMF polyp).

Symptoms of leiomyoma:

One or more of the following symptoms maybe present in **multiple** or **large** fibroids.

1. *Abnormal uterine bleeding (AUB):*
   See before.
2. *Pelvic pain:*
   Fibroids are **painless unless complicated**.
   Types of pain:
   A. Dull-aching pain:
      In hyaline degeneration and infection of SMF polyp.
   B. Acute abdominal pain:
      In red degeneration and torsion of pedunculated SSM.
   C. Colicky pelvic pain:
      With extrusion of SMF polyp through the cervix.
   D. Dysmenorrhea (congestive or spasmodic):
      With SMF and SMF polyp.
   E. Pain due to common associations:
      As endometriosis.
3. *Pressure symptoms:*
   A. Pressure on urinary bladder → frequency of micturition and dysuria.
   B. Pressure on urethra by large cervical leiomyoma → urine retention.
   C. Pressure on pelvic nerves → referred back and thigh pain.
4. *Infertility:*
   - Associated cause of infertility in only **5-10%** of cases.
   - Sole cause of infertility in less than **3%** of cases.

   Mechanism:
   a. Interference with implantation due to distortion of uterine cavity as in SMM.
   b. Tubal obstruction by multiple ISM or bilateral cornual fibroids.
   c. Interference with sperm motility through the cervical canal by large cervical fibroids.
5. *Other symptoms:*
   A. Pregnancy complications:
      a. Pregnancy:
      RPL, PTL
      b. Parturition:
      Dysfunctional/prolonged (atony and malpresentations), postpartum hge (atonic, traumatic, retained placenta)
      c. Puerperium:
      Sepsis due to bleeding, prolonged labor
      d. Placental complications:
      Accreta and accidental hge
      e. Ectopic pregnancy:
      If causing tubal stretch and stenosis
   B. Leucorrhea:
      Due to pelvic congestion or infected fibroid polyp (+ foul discharge).

C. *Progressive abdominal enlargement:*
Huge myomas arise in pelvis and rise gradually above pelvic brim (± pressure symptoms).

---

3. Indications to operate on symptomless fibroid.

Indications to operate on asymptomatic myomas:

1. Uterus larger than 14 weeks size (will cause unpleasant pressure symptoms).
2. Pedunculated SSM (liable to torsion).
3. Cervical or BLMs (possible ureteric compression if they enlarge).
4. Rapid growth and rapid recurrence after myomectomy (suspicion of malignancy).
5. If diagnosis is doubtful (ovarian origin is suspected rather than uterine mass).

---

4. Types, indications, contra-indications and complications of myomectomy.

Definition:

- Surgical procedure in which myomas are enucleated from their bed in the myometrium, while preserving the whole uterus for further menstruation and child-bearing.
- More appropriate for young patients with infertility or low parity.
- Surgical removal of leiomyoma alone (myomectomy) or with the uterus (hysterectomy).
- The only definitive treatment of uterine fibroids.

Indications of surgical treatment:

1. Uterine size > 14 weeks pregnancy size (due to multiple or large myoma).
2. Severe AUB causing severe anemia affecting the patient's wellbeing.
3. When myoma is proved to be responsible for infertility or recurrent pregnancy loss.
4. SMF polyp protruding from the cervix (due to infection, infertility and foul discharge).
5. BLMs and cervical myomas are better removed since their growth may lead to ureteric compression and serious back pressure effect on kidney and ureter.

Contra-indications:

1. <u>During pregnancy:</u>
   - Increased uterine vascularity and poor contractility → excessive bleeding and increased risk of abortion.
   - Torsion in pedunculated SSM is an exception (emergency myomectomy).
2. <u>After menopause:</u>
   Myomas that present by increase in size or post-menopausal bleeding (PMB) are highly suspicious for malignancy → hysterectomy is the only choice when surgery is indicated.
3. <u>Suspicion of sarcomatous changes:</u>
   a. Rapid growth without evidence of degeneration.
   b. Tumors that first appear or increase in size in menopause.
   c. Tumors that present with post-menopausal bleeding (PMB).
   d. Rapid recurrence after surgical excision.
4. <u>Multiparous perimenopausal women:</u>
   No point for preserving uterus with chances of recurrence.
5. <u>Multiple huge myomas regardless age and parity:</u>
   Myomectomy is difficult and associated with marked blood loss (may endanger patient's life).

Types (according to the route):

Operation	*Indication*	*Advantages*	*Disadvantages*
1. Abdominal myomectomy (the commonest approach)	**<u>Operation of choice in multiple and large fibroids.</u>**		Opening uterine cavity in ISM is considered a risk factor for future uterine rupture (indication for cesarean section in subsequent pregnancies).
2. Vaginal myomectomy	For **pedunculated SMF polyp** in endometrial cavity or **protruding through the cervix.**		
3. Hysteroscopic myomectomy	For **small SMM < 5.0 cm** in diameter which **protrude > 50%** in uterine cavity.	Minimal pain and bleeding with short recovery.	
4. Laparoscopic myomectomy	**<u>When myomas are:</u>** • Limited to < 4 in number. • < 6 cm in size. • Mainly SSM (rarely ISM). • Uterus is < 16 weeks size.	• Less postoperative pain. • Shorter and better postoperative recovery period.	• Limited indications. • Longer operative time. • Need for advanced instruments and well-trained team.

Complications of myomectomy:

1. *Immediate complications:*
   A. <u>Excessive blood loss during operation:</u>
      - Due to increased vascularity and inadequate hemostasis.
      - <u>Minimized by:</u>
        - Use of intra-operative tourniquet → to compress uterine and ovarian vessels.
        - Or injection of intra-myometrial vasopressin → vasoconstriction.
        - Or use of GnRH agonists (3-6 months preoperatively) → diminish size and vascularity of myomas.
        - Timing of surgery (post menstrual).
        - Single midline incision.

   B. <u>Increased incidence of postoperative low grade fever, anemia, pain and ileus.</u>

2. *Delayed complications:*
   A. *Persistent symptoms:*
      As menorrhagia, dysmenorrhea and infertility esp. in case of large myoma and associated adenomyosis.
   B. *Recurrence of fibroid (27% of cases):*
      More common in the following cases:
      - After removal of multiple and large fibroids in younger patients.
      - High-risk population (dark races, African women, +ve family history).
      - Incomplete removal of fibroids at primary surgery leaving seedling small myomas to grow by time esp. in young patients.
   C. *Postoperative pelvic and peritubal adhesions:*
      Leading to tubal factor of infertility and rarely intestinal obstruction.
   D. *Rupture of uterine scar:*
      During labor or rarely during pregnancy.
3. *General complications of surgery:*
   Anaesthesia, DVT, preeclampsia, keloid and hernia

---

5. Discuss treatment options of fibroid.

1. *Conservative approach:*
   - Observation with periodic follow up: in small asymptomatic cases.
   - 6-months intervals of follow up.
   - During pregnancy, all myomas are managed conservatively until after puerperium.
   - *Indications to operate on asymptomatic myomas:*
     See before.
2. *Medical treatment:*
   Aim:
   Control symptoms associated with small myomas (AUB, dysmenorrhea & pelvic pain).

Drug	*Mechanism*	*Uses*
NSAIDs	Inhibit PGL synthesis and restore PGL/prostacyclin ratio.	- Reduce menstrual flow. - Treat dysmenorrhea and pelvic pain.
Progestins	Synthetic gestagens induce pseudo-decidual and atrophic changes in the endometrium.	- Control mild bleeding. - Induce regular endometrial shedding when used on cyclic bases (see ttt of DUB).
GnRH agonists	Such as Leuprolide acetate *(3.75 mg IM monthly for 3-6 months)* **suppress gonadotropin secretion** and create **hypo-estrogenic state** (pseudo-menopause) → 2ry amenorrhea with temporary reduction in size and vascularity of fibroids.	- Is **temporary, expensive** with **recurrence** to initial size in 12 weeks of stopping therapy. - But, associated with side effects of estrogen insufficiency (**menopause-like**

		symptoms).

3. Surgical treatment:

a. <u>Myomectomy:</u>

Either vaginal (polypectomy) or abdominal

b. <u>Endoscopic:</u>

Vaginal (hysteroscopy) or abdominal (laparoscopy)

c. <u>Hysterectomy:</u>

Vaginal (if with prolapse), abdominal or laparoscopic / may be total, subtotal or bilateral with salpingo-oophorectomy

d. <u>New methods:</u>

Myolysis, embolization or MRI guided focused US

---

6. Discuss diagnosis and treatment of fibroid (June 2000)

Diagnosis:

Clinical picture:

Symptoms:

See before.

Signs:

1. General examination:

- No specific signs.
- Pallor if anemia is present.

N.B. It may be associated with polycythemia not anemia

2. Abdominal examination:

- Large myoma are felt abdominally if the uterus enlarges above the pelvic brim as:
  - Central firm mass.
  - Bossy irregular contour.
  - Usually not tender.
  - Limited mobility.
  - Dull on percussion.
- Upper border is felt by ulnar border of left hand (may reach up to umbilicus or higher).
- Lower border is usually not felt by the other hand as the mass is pelvi-abdominal.

3. Local examination:

A. <u>PV examination:</u>

Detect cervical myomas and SMF polyp protruding through the cervix.

B. <u>Bimanual examination:</u>

Detect uterine enlargement (maybe firm with bossy contour, not tender, with restricted mobility).

C. <u>Speculum examination:</u>

Visualize cervical myomas and SMF polyp protruding through the cervix.

Differential diagnosis:

1. Large fibroid → from other causes of pelvi-abdominal swellings.
2. SMM or ISM → from other causes of symmetrical enlargement of the uterus.
3. Calcified myoma → from other causes of calcified pelvic shadow on plain x-ray.

4. SMF polyp → from other causes of a mass protruding from the cervix.

Investigations:

1. *Ultrasonography (US):*

Gold standard in diagnosis:

   a. Assessment of site, size, number and relation to endometrial cavity.
   b. Detection of adnexal pathology and exclusion of pregnancy complications.
   c. Saline infusion sonography (SIS) is sensitive in diagnosis of SMM and SMF polyps.

2. *MRI and CT scan:*

   Diagnostic but not superior to US which is cheaper, easier and more readily available.

3. *Hysterosalpingography (HSG):*
   - Diagnosis of SMM that appears as a filling defect.
   - Testing tubal patency in patients complaining of infertility.

4. *Hysteroscopy:*

   Gold standard in diagnosis of small endometrial polyps, SMF polyps and SMM.

5. *Laparoscopy:*

   Valuable in differential diagnosis of complex adnexal masses.

Treatment:

See before.

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7. Definition and pathology of red degeneration of fibroids. Why is it more common in pregnancy?

Red degeneration (Necro-biosis):

- Commonest change during **pregnancy**, due to:
  - Tumor may outgrow its blood supply.
  - Or kinking of blood vessels due to rotation resulting from rapid uterine growth.
  - Or increased tendency to thrombosis associated with pregnancy.
- Reflects a state of incomplete necrosis (from which the tumor is capable of recovery).
- Thrombosis of capsular vessels → ischemia and release of lipoid toxin → intravascular hemolysis and diffusion of blood pigments giving the tumor red color.
- Clinically:
  - Myomas become enlarged, tender.
  - Acute pelvic pain.
  - Mild pyrexia.
- Management:

  Conservative (rest and analgesics as NSAIDs).

---

8. Pathological changes in fibroid (june 2017).

Pathological changes:

1. *Atrophy:*
   - **Commonest** change occurring in **menopause**.
   - Caused by decreased blood supply secondary to estrogen deficiency.
   - Also occurs during: puerperium and long term treatment with GnRH agonists.
   - Clinically: atrophy is associated with diminished size which is usually **silent** and

painless.

2. *Hyaline degeneration:*
   - Commonest change in **child-bearing period**.
   - Mostly occurs in the **center** of the tumor due to diminished vascularity.
   - Microscopically:
     - Whorled appearance is lost.
     - Tumor tissue is replaced by homogenous structureless material stained pink with eosin.
   - Clinically:
     - Tumor consistency becomes **soft**.
     - If increase in size occurs, it becomes tense cystic.
     - Mild to moderate dull-aching pain.
3. *Cystic degeneration:*
   - **Rare**.
   - Due to absorption of liquefied hyaline material at the center of the myoma.
   - Microscopically: cavity is lined by remnants of unabsorbed hyaline material.
   - Clinically:
     - Tumor becomes **soft** and **cystic**.
     - Mild dull-aching pain.
4. Red degeneration : see before .
5. *Fatty degeneration:*
   - Deposition of fat within the muscle cells.
   - It's the precursor of calcification.
6. *Calcification:*
   - Common in **long-standing myoma** and in **menopause**.
   - Caused by deposition of calcium phosphate and carbonate along the blood vessels (**hard** consistency).
   - It is either peripheral (egg shell) or diffuse (womb stone appearance on plain x-ray or US).
7. *Necrosis:*
   - Commonly occurs at the **tip of SMF polyp** due to poor blood supply.
   - Also occurs following **torsion of pedunculated SSM**.
8. *Infection:*
   - Occurs at necrosed tip of SMF polyp projecting into uterine cavity, cervix or vagina.
   - May occur during puerperium, or as spread from nearby infected organs as appendicitis.

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9. Cervical polyps.

1. *Mucous polyp:*
   - Origin: one or more reddish soft polyps seen within the endocervix.
   - Etiology: hyperplasia of endocervical epithelium due to chronic cervicitis.
   - Diagnosis: speculum examination reveals the lesion by naked eye, or hysteroscopy.
   - Treatment: polypectomy, followed by treatment of chronic cervicitis.
   - N.B. Any polyp removed should be examined histologically to exclude malignancy.

2. *Fibro-adenomatous polyp:*
   - Mucous polyp in which the stroma is dense and fibrous.
3. *Fibroid polyp:*
   - Rare.
   - Appears as firm polyp with necrosed tip attached to cervix by short pedicle.
   - Treated by vaginal polypectomy.
4. *Malignant polyp:*
   - Carcinoma, sarcoma or rare highly malignant grape-like sarcoma of children (mixed cell sarcoma) characterized by rapid invasion and formation of grape-like mass of soft pinkish edematous polyp which fill upper vagina.
5. *Bilharzial papilloma:*
   1. Usually develop from the vaginal surface of cervix.
   2. Single or multiple.
   3. Sessile or pedunculated.
   4. Variable sizes.
   5. Firm consistency.
   6. Usually rough and covered by intact greyish pink mucous membrane.

Other topics:

1. Etiology of leiomyoma.

Risk factors:

1. Parity:
   More common in nulliparous and low parity women.
2. Hereditary factors:
   More in women with positive family history (mother and sister).
3. Racial factors:
   More common in dark races.
4. Obesity:
   More common in women with higher body mass index (BMI).

Common associated conditions:

1. Follicular cysts of the ovary.
2. Endometriosis and adenomyosis.
3. Endometrial hyperplasia.
4. Rarely, endometrial carcinoma.

Etiologic factors:

Exact etiology is unknown.

1. *Abundant estrogen environment (estrogen-dependent tumor):*
   Evidences:
   a. Leiomyomata appear only in child-bearing period.
   b. No new tumors develop before puberty or after menopause.
   c. Increase in size during pregnancy and estrogen therapy.
   d. Decrease in size and atrophy during menopause or GnRH agonist therapy (estrogen deficiency).
   e. Commonly associated with endometrial hyperplasia and endometriosis.
2. *Growth factors:*
   a. Increased production of Epidermal Growth Factor (EGF).
   b. Decreased production of Growth Inhibiting Factor (GIF).
   N.B. Both may act synergistically with E_2 to induce growth of myoma.
3. *Genetic factors:*
   40% of cases show cytogenetic abnormalities, suggesting gene mutation.

---

2. Pathology of leiomyoma.

1. *Site:*
   A. *Corporeal leiomyoma:*
   - 95%.
   - Develop within myometrium of uterine body.
   - Usually multiple and of variable size.
   - Classification according to relation to endometrium (uterine mucosa):
     a. Interstitial myoma (ISM):
        Within the center of myometrium.
     b. Subserosal myoma (SSM):

- Raise the peritoneal covering of uterus (serosa), externally.
- May acquire a pedicle (pedunculated SSM), or burrow within the leaves of the broad ligament (2ry broad ligamentary myoma BLM).

c. Submucosal myoma (SMM):

- Indent the endometrial lining and encroach on endometrial cavity.
- May acquire a pedicle (pedunculated SMM).

B. Cervical leiomyoma:

- < 5%.
- Usually solitary.
- From portio-vaginalis:
  - Grow downward and project into vagina.
  - May reach the introitus and protrude outside vulva.
- From supra-vaginal cervix:
  - Grow upwards in the true pelvis in relation to urinary bladder, ureters, rectum and broad ligament.

C. Broad ligament myoma (BLM):

- Rare (< 1%).
- 1ry BLM (True BLM):
  - Arise from muscle fibers within the broad ligament.
  - Not connected to the uterus.
- 2ry BLM:
  - SSM that grow externally from the side of the uterus to burrow within the anterior and posterior leaves of the broad ligament.
  - Connected to the uterus directly or through a pedicle.

2. Size:

Variable (ranging from size of pea-nut to size of fetal head).

3. Shape:

Rounded or spherical.

4. Number:

- Corporeal leiomyomas are usually multiple.
- Cervical and BLM are usually single.

5. Consistency:

- Firm.
- If degenerated, becomes soft (hyaline degeneration), cystic (cystic degeneration) or hard (calcification).

6. Cut section:

- Well-circumscribed tumor.
- Whorled appearance: due to interlacing bundles of muscle cells with fibrous tissue.
- Paler than rest of myometrium: due to poorer vascularity.
- Pseudo-capsule: formed of compressed normal myometrium (does not belong to the tumor).
- Blood vessels: lie in the capsule and send radial branches to the center → so calcification occurs in the periphery while degeneration occurs at the center (poorer blood supply).
- Fibroid polyp: SMF acquires a pedicle and protrudes into the endometrial cavity forming

SMF polyp → capsule usually ruptures and retracts (fibroid polyp receives blood supply from vessels in the pedicle).

7. *Microscopic picture:*
   - Smooth muscle cells arranged in bundles interlaced with fibrous connective tissue.
   - Vascular structures are few.
   - Mitosis is rare.
   - Cellular leiomyoma: tumors with mitotic counts of 5-10 per 10 consecutive high power field (HPF) but lack cytological atypia (not considered malignant).

---

3. Complications and effects of leiomyoma.

Complications:

1. *Torsion* of pedunculated SSM or rarely torsion of the whole uterus.
2. *Rupture* of a surface vein of SSM → intraperitoneal hemorrhage.
3. *Incarceration* or impaction of SSM in the pelvis esp. during pregnancy.
4. *Malignant transformation into leiomyosarcoma:*
   - Incidence: very rare (0.2-0.5%).
   - Suspected in case of:
     a. Rapid growth without evidence of degeneration.
     b. Tumors that first appear or increase in size in menopause.
     c. Tumors that present with post-menopausal bleeding (PMB).
     d. Rapid recurrence after surgical excision.
   - Gross picture (during surgery):
     – Yellowish in color.
     – Friable.
     – Areas of hemorrhage and capsular invasion → difficult enucleation (not capsulated).
   - Microscopic picture:
     Mitotic count: 10 mitotic figures per 10 HPF + frequent cellular atypia & coagulative necrosis.

Effect of leiomyoma on uterus and pelvic organs:

1. Uterine enlargement (symmetrical or asymmetrical according to site).
2. Distortion of endometrial cavity (in SMM and SMF polyp).
3. Displacement in uterine position in large myomas (fundal, BLM and cervical).
4. Increased vascularity and venous congestion (in multiple and large myomas).
5. Pressure on urinary bladder → frequency of micturition.
6. Overstretching on urethra by large cervical myoma → retention of urine.
7. Compression on ureters by large cervical and BLMs → ureteric back pressure → hydroureter and hydronephrosis.

---

4. Hysterectomy.

Definition:

- Surgical removal of uterus together with leiomyomas.
- The only definitive treatment for leiomyoma that **nullifies chance of recurrence.**

Indications:

Multiparous perimenopausal and menopausal patients esp. when multiple and large or whenever

malignancy is suspected (+ discuss indications of surgical management of leiomyoma in general).

Types and routes:

1. Abdominal hysterectomy:
   - Most popular route (most cases are large multiple myomas).
   - Maybe:
     a. Total hysterectomy:
        Cervix removed with uterus.
     b. Subtotal hysterectomy:
        Cervix is left attached to vaginal vault.
2. Vaginal hysterectomy:
   Preferred when uterus is small, esp. in presence of any degree of pelvic organ prolapse.
3. Laparoscopic hysterectomy:
   Limited use except when myomas are small in size.
4. Laparoscopic-assisted vaginal hysterectomy:
   Facilitates the vaginal procedure by ligating the infundibulo-pelvic ligaments laparoscopically prior to vaginal hysterectomy.

Techniques:

See operative gynecology chapter.

Advantages over myomectomy:

1. Sure relief of symptoms with no chance of recurrence.
2. Less blood loss during surgery " bloodless operation " .
3. Lower postoperative morbidity.

Ovarian conservation during hysterectomy:

Ovaries should be preserved in young patients with healthy ovaries and negative family history of ovarian and breast cancer to maintain their function and avoid early use of hormone therapy.

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5. Other therapeutic options in management of leiomyoma.

- Alternatives to traditional surgery especially in cases unfit for or refraining from surgery.
- Most are still not standardized although promising results are reported.

1. Uterine artery embolization (UAE):

Procedure:

Uterine artery or its branches are occluded by injection of gel foam pledgets, through a catheter introduced via femoral artery → depriving tumor from its main feeding vessels → shrinkage in size, atrophy and necrosis.

Advantages:

- Relatively safe.
- 60% reduction in size and 90% control of menorrhagia in 8-12 weeks.

Disadvantages:

- Significant postoperative pain (like red degeneration).
- Need for skillful radiology team.
- Limitations related to the size and number of myomata that could be managed successfully by this procedure.
- May not be suitable for patients seeking future fertility.

2. *Laparoscopic myolysis:*

Procedure:

Laparoscopic myolysis using laser or coagulation current or cryo-myolysis using -180 C probe.

Advantage:

Persistent decrease in size of myomas.

Disadvantages:

- Effective only in small and SSM which are mostly asymptomatic.
- More studies needed to substantiate its role in management of leiomyoma, patient selection and long term adverse effects.

3. *MRI-guided focused ultrasound:*

Procedure:

- Relatively new treatment option that uses ultrasound generated heat to cause cell death.
- Fibroids are localized by MRI and heat from a phased array ultrasound transducer is directed towards the myoma to produce protein denaturation.

Advantages:

Decrease in size and improvement of symptoms in up to 60% of cases.

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6. Uterine polypi.

1. *Corporeal polypi:*

A. *Adenomatous endometrial polyp:*

Origin:

From endometrium, either as single or multiple adenoma in association with marked endometrial hyperplasia (EH).

Symptoms:

a. Abnormal uterine bleeding (AUB):
   - Menorrhagia.
   - Metrorrhagia.
   - Postcoital bleeding.

b. Leucorrhea:

   Polyp maybe associated with excessive vaginal discharge.

Signs:

If protruding through the cervix, it appears by speculum examination as:

- Tongue-like projection.
- Soft consistency.
- Flat compressed tip.

Diagnosis:

All the following are efficient:

- TAS.
- TVS.
- SIS.
- 3D US.
- Hysteroscopy.

Treatment:

Polypectomy followed by hormonal treatment.

Polypectomy is done via:

a. Hysteroscopic-guided polypectomy (**gold standard treatment**).
b. Dilatation, polypectomy and curettage (D & C), if hysteroscopy is not available.

N.B.

Histopathologic examination of the polyp is important to confirm diagnosis.

B. *SMF polyp:*

Origin:

Myometrium of body of uterus, as a SMM that acquires a pedicle and protrudes into endometrial cavity.

Symptoms:

- AUB, menorrhagia, metrorrhagia.
- Foul discharge.

Signs:

If protrudes through cervix, appears by speculum examination as:

- Rounded mass.
- Firm consistency.
- Necrosed infected tip.
- Long pedicle.

Treatment:

- Hysteroscopic-guided polypectomy (**gold standard treatment**).
- Occasionally, a large SMF polyp > 6 cm will require abdominal myomectomy in young patients, or hysterectomy in perimenopause.

C. *Placental polyp:*

Origin:

Accumulated blood clots over the surface of retained placental fragment.

Symptoms:

Persistent bleeding after labor or abortion.

Signs:

Subinvolution of uterus with softer consistency.

Diagnosis:

All the following are efficient:

- TVS.
- TAS.
- SIS.
- 3D US.
- Hysteroscopy.

Treatment:

- Hysteroscopic-guided polypectomy (**gold standard treatment**).
- D & C polypectomy, if hysteroscopy is not available.

N.B.

Microscopic examination is important to exclude choriocarcinoma.

D. Malignant polyp:

- Carcinoma.
- Sarcoma.
- Chorionepithelioma.

2. Cervical polypi:

See before.

Endometriosis & Adenomyosis

1. Surgical treatment of adenomyosis (sep 2012).

Hysterectomy:

- Treatment of choice in perimenopausal bleeding not responding to medical or hormonal treatment
- The only way to establish diagnosis with certainty

"Discuss hysterectomy"

---

2. Adenomyosis uteri (sep 2011)

Definition:

Presence of endometrial glands & stroma deep within the myometrium.

Aetiology:

Unknown but theories postulate that it may be due to growth of endometrial glands deeply between muscle fibers of myometrium in presence of estrogen environment
associated with smooth muscle hypertrophy around the ectopic glands.

Pathology:

A. *Diffuse type:*
   Symmetrically enlarged, globular in shape and firm in consistency reflecting myometrial thickening generally affecting fundus but may also involve either or both uterine walls.
B. *Localized type:*
   Similar to leiomyomata except that adenomyosis is not encapsulated
   the cut section may have whorl like or grandular trabecular pattern with small yellow or brown cystic spaces containing fluid or blood.
   "Both adenomyosis & fibroid may coexist in the same uterus (adenomyoma)"

Diagnosis: (= clinical picture including symptoms, signs, complications & D.D. + investigations)

Symptoms:

1. Secondary spasmodic dysmenorrhea due to contractions provoked by premenstrual swelling & menstrual bleeding
2. Menorrhagia due to increased uterine size & endometrial surface area

Signs:

Symmetrically enlarged uterus which is sometimes tender especially in the premenstrual period (Halban's sign)

D.D from other causes of symmetrically enlarged uterus:

1. Pregnancy & its complications
2. SMM
3. Endometrial adenocarcinoma
4. Pelvic congestion syndrome (Taylor syndrome) = chronic pelvic pain + menometrorrhagia
5. DD of pelvi-abdominal swellings - see p 270, 271 in department book.

Complications:

1. Anemia
2. Primary adenocarcinoma

Investigations:

1. MRI:
   Investigation of choice as it gives excellent image of myometrium with areas of adenomyomas
2. Ultrasonography (TAS/TVS/3DUS):
   Will reveal thickened myometrium, sometimes associated with localized areas of different echogenicity similar to leiomyoma

Treatment options:

1. Dysmenorrhea can be managed by NSAIDs.
2. Menorrhagia can be managed by:
   - Antifibrinolytics as tranexamic acid & venotonics as Diosmin
   - Gestagens as norethisterone acetate 5 mg t.d.s. for 2w/months
   - Combined estrogen/progesterone as COCs 21 days/months
   - LNG IUD (Mirena) effective in control of bleeding within 3 months of insertion

[A side note: Hormonal treatment usually fails because the basal layer of the endometrium is hormone insensitive]

3. Hysterectomy (see Q1).

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3. Diagnosis of endometriosis (sep 2014).

Clinical Picture = (symptoms, signs, DD, complications, investigations)

Symptoms:

- **Asymptomatic** (endometriosis is accidentaly discovered during laparotomy or laparscopy).
- If symptomatic →pain &/or infertility
  1. *Pain:*
     (**6 Ds**: 3 main: **D**ysmenorrhoea-**D**yspareunia-**D**eep pelvic pain
     + 3 others: **D**ysuria-**D**yschezia-**D**orsal back pain)
     a. *Dysmenorrhea :*
        - One of the commonest clinical presentation.
        - Pain starts few years after a period of normal non-painful menstrual cycle.
        - Starts during menstruation &increase rapidly due to distension of ectopic endometrial glands with blood without having an exit.
        - Pain decreases gradually towards the end of the cycle due to absorption of blood.

     b. *Chronic pelvic pain:*
        Diffuse or localized chronic pelvic pain > 6months is strongly suggested for endometriosis.

     c. *Dyspareunia:*
        In case of endometriosis dyspareunia is 2ry to endometriotic implants of the uterosacral ligaments, Douglas pouch, ovarian endometriosis or due to associated fixed RVF uterus.

     Mechanism of pain in endometriosis :

     - Distension of ectopic endometrial gland in a closed space with blood having no excit.
     - Release of inflammatory mediators from superficial lesions.
     - Irritation of pelvic nerves or involvement by adhesions in deep lesions.

2. *Infertility:*
    - Endometriosis will be discovered at laparoscopy up to 30% of infertile females however in many cases it may be association rather than a cause.
    - Moderate to severe endometrosis: may compromise fertility thought creating pelvic adhesions, which may be peritubal or periovarian ,impairing ovum pick up.
    - Mild endometriosis: may affect fertility through inducing luteal phase defect& increased tubal phagocytic macrophage activity on sperms.
3. *Other Symptoms:*
    - GIT symptoms: pain during defection (dyschezia), intestional cramps & rarely cyclic rectal bleeding.
    - Urinary symptoms: dysuria, frequency &rarely cyclic hematuria.
    - Distant metastasis: symptoms according to organ involved.

Signs:

1. *General examination:*

    No general specific signs.
2. *PV & Bimanual examination (only in extensive conditions):*
    - The uterus may be fixed in AVF or RVF position.
    - Tender palpable nodules on uterosacral ligaments &pouch of Douglas.
    - Endometriosis (chocolate cyst):

      May be palpated as tender tense cystic fixed adnexal masses in non-obese patients.

Investigations:

1. *Laparoscopy:*
    - **Gold standard** in diagnosis & staging
    - Indicated in: chronic pelvic pain, infertility & unresolved adenxal masses.
    - Laparoscopy allows direct visualization of endometrotic implant & performing biopsies for confirming direct visualization:
        - Typical lesion: endometrotic implant appears as brown or black pigmentations (powder brown lesion) on peritoneal surfaces, surrounded by adhesions.
        - Atypical lesions: clear vesicles, white opacified or red hemorrhagic polypoid excrescences
2. *Ultrasonography & MRI:*
    - Small endometriotic lesions are not visualized with pelvic us.
    - MRI may detect large implants and deep seated ones.
    - Both US & MRI are sensitive indiagnosis of endometriomas even if small in size.
3. *CA-125:*
    - Cells release Ag of coelomic epithelium (Normal 5-53μml).
    - It shows slight to moderate increase in many cases, but not specific.
    - Useful marker for response to ttt in cases with initially elevated levels. (= Of Prognostic value only not diagnostic)

Differential diagnosis:

1. Causes of chronic pelvic pain: PID, irritable bowel syndrome, ureteric pain, cystitis .
2. Endometriomas from other causes of adnexal masses: Hgic ovarian cyst, tube-ovarian complex & pelvic malignancy.
3. Fixed RVF causes.

4. Discuss treatment option of a 28 year old patient with diagnosis of pelvic endometriosis and right ovarian endometrioma of 5 cm & pelvic pain and dyspareunia (June 2017)

1. Medical treatment:

	Combined Oral Contraceptive	**Synthetic Progesterone (Gestagens)**	**Long Acting GnRH Agonists (e.g. Leuprolide)**
Aim	Induce amenorrhea with a **pseudopregnency** state		Induces amenorrhea with a **pseudomenopausal state**
Mechanism	Amenorrhea, Pseudo-decidualization & atrophy of ectopic endometrium		- Menopause like hypoestrogenic state with atrophy of ectopic endometrium - It starts with an initial FSH stimulation followed by down regulation of receptors with suppression of FSH & LH & consequently estrogen.
Dose	Continuous COCs once daily for 4-6 months.	Continuous gestagens treatment for a 6-9 months course: - Norethisterone acetate: 5 mg orally 2-3 times daily - Dienogest: 2 mg oral tablets, once daily - Depot medroxy progesterone acetate (DMPA): 150 mg injections every 3 months	Depot forms given IM monthly or every 3 month for 6-9 months.
Disadvantages	Side effects of hormone therapy as weight gain, bloating, headache	Side effects as bloating, weight gain, headaches & **breakthrough bleeding.**	- Symptoms of menopause as hot flushes, vaginal dryness, accelerated bone loss - Adverse effects can be minimized by the use of low dose estrogen.

2. *Surgical treatment:*

Conservative surgery:

a. Excision,fuguration or laser ablation of visible lesions.
b. Lysis of pelvic peritubal & periovarian adhesions.
c. Excision of the endometrioma (since it is > 4 cm)

Conservative surgery can be achieved via :

a. Laparoscopy:

- It is the gold standard
- Suitable for infertile patients aiming to restore normal anatomy with creating **minimal postoperative adhesions.**

b. Laparotomy:

- For severe cases with **extensive adhesions or large endometrioma**

N.B.

Preoperative hormonal treatment by 3 months of GnRH may reduce vascularity & nodular, size,facilitating surgery & improving the prognosis.

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5. Treatment of pelvic endometriosis (June 2014, 2016).

Planned accourding to age of the patient, severity of the symptoms, future reproductive plans & extent of disease at laparoscopy.

1. *Expectant management:*
   - For young patients with **minimal symptoms & signs.**
   - The goal of expectant management:
     - Control of pain using **NSAIDs**
     - **Follow up** for growth of lesions
2. *Medical treatment:*
   - For patients with **marked symptoms & minimal signs**
   - Treatment is based on the observation that symptoms of endometriosis improve during pregnancy & menopause (See previous Q)
3. *Surgical treatment :*

   Surgery is the best choice in cases with pelvic adhesion &larger endometriosis.

   a. *Conservative surgery :*

   See previous Q

   b. *Extirpative surgery:*
      - TAH & BSO in the procedure usually performed in elderly patients, with extensive adhesions and no desire for fertility.
      - Estrogen replacement therapy is recommended in young patients to relieve associated post menopausal symptoms.
      - ERT carries only a small risk of recurrence while if the ovaries were preserved the recurrence rate will be high with 15-40% chance to re-operate.

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6. Discuss management of endometriosis in infertile patients (sep 2016).

Introduction:

Endometriosis is one of the major causes of infertility, 30%. It mainly causes infertility by

affecting tubal factor, because of extensive adhesions. (Mild endometriosis may lead to increased macrophage spermicidal action. Dyspareunia experienced by patients also plays a role in decreased feritlity in patients.) Associated pathologies may affect fertility in a patient with endometriosis as fibroids - they should be treated as well.

Treatment should be done according to laparascopic findings:

1. *Conservative laparoscopic surgery:*
    - Diagnosis & staging of endometriosis
    - Ablation of endometriotic implants & lysis of adhesions
    - Excision of large endometriomas > 4 cm
    - Evaluation of tubal & peritoneal factors of infertility
2. *Suppression of menses:*

Using GnRH agonists for 6 months postoperative.

OR

3. *Active management of infertility via:*
    - Controlled ovarian stimulation
    - ART: IUI & ICSI

Steps of ICSI:

1. Pituitary down regulation: using GnRH agonists to prevent natural LH surge
2. Ovarian stimulation: using daily FSH injection to induce follicular growth and maturation
3. Ovulation trigger: by hCG to induce artificial LH surge
4. Oocyte retrieval: 24 hours after hCG trigger
5. Lab sperm-egg fertilization: sperm is injected directly into cytoplasm of oocyte
6. Embryo transfer: 2-3 days after oocyte retrieval
7. Luteal phase support: for 2 weeks by progesterone to avoid premature shedding of endometrium

5. PELVIC FLOOR DISORDERS

Prolapse

1. Degrees of uterine prolapse. (1998)

In uterine prolapse descent of the uterus is judged by the level of the cervix, where the external cervical os normally lies at the level of the ischial spines.

- **1st Degree:**

  The cervix descends downwards through the vagina, but the external os does not reaching the introitus (hymenal ring).
- **2nd Degree:**

  The external os reaches or slightly protrudes outside the introitus.
- **3rd Degree:**

  The whole uterus except the fundus descends outside the introitus.
- **Procidentia:**

  The whole uterus including the fundus descends outside the introitus.

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2. Discuss causes, types, symptoms, DD & treatment of prolapse. (June 2003)

Causes:

i. Predisposing Factors:

1. Childbirth trauma:

Damage to the pelvic supports during vaginal birth trauma is the most frequently encountered risk factor for pop. This might be due to overstretch of the cellular tissue and damage to the muscular innervations. It is aggravated by the following conditions:

- Difficult labour with oversized fetus and/or prolonged 2nd stage
- Short interval successive deliveries without appropriate involution periods.
- Direct pelvic floor injury as in perineal lacerations without prompt repair

2. Menopausal atrophy:

Older menopausal women are at higher risk for POP due to degenerative changes in pelvic supports secondary to prolonged estrogen deficiency. Risk increases by increased age.

3. Congenital weakness:

POP may occur in some women in a young age and in absence of childbirth trauma. This will mostly attributed to congenital weakness in pelvic fascia, ligaments, and muscles. It may be associated with poor innervations (as in spina bifida), or generally poor mesenchyme with as associated abdominal wall hernia, rectal prolapse, and or visceroptosis

4. Iatrogenic weakness:

This might be induced by improper technique for supporting vaginal vault during abdominal or vaginal hysterectomy, hence increasing the risk for vaginal vault prolapse.

ii. Precipitating Factors:

Chronically increased intra-abdominal pressure as occurring in chronic cough and constipation may act as an important precipitating factor for POP, especially in elderly

high risk women. And unless properly managed it will act as a major cause for recurrence of the prolapse after surgical treatment.

Types:

i. Vaginal prolapse:

1. **Anterior compartment:**
   - Urethrocele: descent of urethra + lower 1/3 of vagina.
   - Cystocele: descent of the urinary bladder upper 2/3 of anterior vaginal wall
   - Cysto- urethrocele: full vaginal length bladder and urethra
2. **Posterior compartment:**
   - Rectocele: middle 1/3 of posterior vaginal wall + rectum
   - Enterocele: upper 1/3 of posterior vaginal wall small bowel loops
3. **Central compartment (Apical):**
   - Utero-vaginal prolapse: uterine descent with inversion of the vaginal apex
   - Vault prolapse: descent of the blind ended vaginal apex after hysterectomy

ii. Uterine prolapse:

In uterine prolapse descent of the uterus is judged by the level of the cervix, where the external cervical os normally lies at the level of the ischial spines.

- 1st Degree: The cervix descends downwards through the vagina, but the external os does not reaching the introitus (hymenal ring).
- 2nd Degree: The external os reaches or slightly protrudes outside the introitus.
- 3rd Degree: The whole uterus except the fundus descends outside the introitus
- Procidentia: The whole uterus including the fundus descends outside the introitus.

Symptoms:

In general mild cases with minimal degrees of utero-vaginal prolapse are asymptomatic. The more severe degrees of POP may be associated with one or more of the following symptom;

1. Sensation of pelvic ***heaviness***; especially towards the end of the day that or disappears by rest.
2. A ***mass*** filling the vagina or protruding from the introitus; that may be felt by the patient on straining or squatting, and disappears by lying down on the back, or by reduction.
3. Low ***backache***; is common in cases of uterine prolapse. Pain is dull aching, or dragging, that radiates to the thighs and legs. It is stimulated by prolonged walk, hard work, or heavy weight lifting. Pain is usually relieved or decreased by rest and lying down on the back. Pain is explained by marked traction on utero-sacral ligaments by the prolonged uterine descent.
4. ***Urinary*** symptoms: are common in the presence of a cystocele, as
   - Frequency of micturition by day; due to mechanical irritation of the trigone.
   - Frequency may develop by night (nocturia); due to residual urine cystitis.
   - Stress urinary incontinence SUI (escape of urine during cough or straining)
   - Inability to complete micturition unless the anterior vaginal wall is reduced upwards and supported by the patient's fingers, to empty residual urine in a bladder pouch.
5. ***Rectal*** symptoms: as heaviness and difficulty when trying to defecate in cases of rectocele.
6. ***Pelvic congestion*** symptoms: in the form of Dysmenorrhea and Leucorrhoea.

Differential Diagnosis:

1. Cystocele and Rectocele: are differentiated from cysts of anterior and posterior vaginal walls, as inclusion dermoid cysts, which are usually small in size, cystic, tense, slightly tender, and non-compressible or reducible.
2. Urethrocele: should be differentiated from urethral diverticulum, which on compression will

lead to discharge of urine or pus via external urethral meatus.

3. Uterine prolapse: should be differentiated from swellings protruding from the vulva as; large fibroid polyp, and chronic inversion of the uterus.

Treatment:

"Management scheme of prolapse"

1. *Prophylactic*
2. *Active:*
   a. Conservative
   b. Surgical:
      - Vaginal prolapse (anterior and posterior):
        - Anterior colporraphy
        - Posterior colpoperineorraphy
        - Classical repair
        - Culdoplasty
      - Vaginal vault prolapse:
        - Abdominal → sarcocolpopexy
        - Vaginal → sacrospinous fixation
      - Uterine prolapse:
        - Mild:
        - Severe:
          Old: vaginal hysterectomy
          Young: abdominal sling
      - Others:
        - Recurrent: mesh
        - Old and not fit: Le fort

"Treatment in details"

The choice of treatment for genital prolapse depends on several factors including:

- Type and degree of prolapse
- Desire for future pregnancies
- Need for preservation of coital function
- Acceptance and readiness for surgical treatment.

1. *Non surgical measures:*
   a. Pelvic floor exercise (Kegel exercises): may delay the need for surgery in mild cases
   b. Pessary treatment is only a temporary option aiming at temporary reduction of the prolapsed organs until the patient is ready for surgery, or in cases where surgical intervention poses unacceptable risks on the patient's health.
2. *Surgical treatment:*

   Surgical treatment is the only curative approach for moderate and severe cases,

   a. Anterior colporrhaphy:

   For repair of the anterior vaginal wall and associated cystocele.

   b. Posterior colpoperineorrhaphy:

   For repair of posterior vaginal wall prolapse and associated rectocele

   c. Classical repair:

   - Cases of cysto- rectocele are best managed by anterior colporrhaphy + posterior

colpo-perineo-rraphy.

- Associated degree prolapse is managed by shortening of Mackenrodt's ligament plication in front of the cervix.

d. Manchester (fothergill's) operation:

- Cases of 2" degree uterine prolapse with marked supra vaginal elongation of the cervix are best managed by amputation of the vaginal portion of the cervix and shortening of the Mackenrodt's ligament.
- Associated cysto rectocele is managed by classical repair

e. Vaginal hysterctomy and fascial repair:

Indicated in peri-menopausal and postmenopausal patients with marked uterovaginal prolapsed.

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3. Causes of prolapse. (june 2005, 2016)

See Q2

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4. Diagnosis, treatment without surgical details of prolapse (Sep 2007)

- Diagnosis = (Symptoms + signs + complications + DD)
- Diagnosis of prolapse is **entirely clinical** (no need for investigations except to detect the cause)

Symptoms:

See Q2

Signs:

A. General examination:

Chest and Abdominal examination are essential to rule out causes for chronic increase in intra abdominal pressure, as chronic cough, ascites, pelvic mass etc...

B. Local examination:

Local examination of the prolapsed mass is performed with the patient in the lithotomy position while maximum straining

- **<u>Inspection:</u>**

  To detect:

  - Type of prolapse (vaginal, or utero-vaginal)
  - Degree of uterine prolapse if present (1, 2nd, or 3 degree or procedentia)
  - Presence of skin changes or decubitus ulcers
  - Associated patulous introitus

- **<u>Digital Palpation:</u>**

  - PR examination to test for extent of rectal involvement in rectocele
  - Test for levator ani function and strength by pressing index and middle fingers on perineal body muscles.

  "How can you test clinically levator ani function? (June 2009)"

  - Test for complete procidentia by being able to approximate the index and thumb fingers above the fundus of prolapsed uterus

- **<u>Diagnosis of associated conditions as:</u>**

  - Supravaginal elongation of the cervix: using a uterine sound measure
  - Stress urinary Incontinence (SUI) by asking the patient to cough during

examination to detect involuntary escape of urine through urethra.

- Test for enterocele; via eliciting impulse on cough, and or gurgle sensation at the vaginal vault. Combined & PR examination may add to accuracy of diagnosis.

Differential diagnosis:

See Q2

Treatment:

See Q2

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5. Give Names & principles of 3 operations for treatment of vaginal vault prolapse (june 2009)

Surgical procedures for repair of vaginal vault prolapse:

1. **Abdominal sacro-colpopexy:** the vaginal vault sutured posteriorly to the sacral promontory using non absorbable suture material,via a laparotomy incision.
2. **Vaginal sacrospinous ligament fixation:** the vaginal vault sutured to the sacrospinous ligament at one side via vaginal approach
3. **Vaginal Mesh repairs:** recurrence of prolapse after surgical correction may occur with up to 30% of cases requiring a second operation within 5 years. Recurrence may be due to weak connective tissue even before the original operation was performed. Mesh augmented pelvic floor repair is gaining more interest recently aiming at improving tissue strength and support.
4. **Le Fort's operations (Colpocleisis):** the procedure aims at obliteration of the vagina, except for two small channels on either side through which normal cervical discharges can escape. Rectangular flaps are excised from the anterior and posterior vaginal walls the raw areas are sutured together. The procedure is reserved for the very old, flail, non-sexually active patients, unfit for lengthy surgery procedures.

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6. Anatomical changes associated with marked long standing female genital organ prolapse. (June 2011)

1. Vaginal skin Keratinization: Everted vaginal skin in cases of prolonged vaginal prolapse becomes thickened, and white with keratin, being exposed to air and trauma.
2. Vaginal and cervical ulcerations: may occur at the most dependent part of the vagina or cervix as a result of congestion and circulatory changes rather than friction with the thighs (decubitus ulcers)
3. Cervical hypertrophy: due to chronic congestion and associated cervicitis.
4. Supravaginal elongation of the cervix; due to stretch on the Mackenrodt's ligament
5. Descent of the base of the bladder with stretching of the urethral sphincter, may lead to frequent desire to micturate and associated stress incontinence.
6. Kinking and compression on the ureters; in severe degrees of utero-vaginal prolapse, may cause back pressure changes in the form of hydro-ureter and even hydro-nephrosis.

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7. List symptoms of vaginal wall prolapse (Sep 2016)

See Q2

Perineal lacerations

1. Mention clinical picture "5 clinical signs" of diagnosis of old complete perineal tear.

Clinical picture:

A. Symptoms:

- The wound is healed by granulation tissue leaving an ugly scar with a weak anal sphincter leading in some cases to incontinence for stool or flatus or both.
- Many patients will be able to regain control over the passage of the hard stools, but remain incontinent to flatus and usually complain of persistent leucorrhea.

B. Signs on clinical examination:

- A defect is noted in perineal body, extending to the anal opening.
- If the rectal wall is also torn, the bright red colour of the rectal mucosa is apparent in the lower part of the defect.
- On each side of the anus the small shallow pit is seen in the skin, these 2 dimples indicate the site of the cut retracted ends of the anal sphincter.
- Absence of normal corrugation around the anus except posteriorly.
- A finger introduced in the anus will confirm absence of sphincter control if the patient asked to contract her muscle.

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2. Complications of perineal tear.

Symptoms of old perineal tear.

1. PPH due to bleeding from lacerations.
2. Infection may occur in laceration site (purple sepsis).
3. Patulous vaginal introitus with unsatisfactory sexual function.
4. Incomplete tear may predispose to gentle prolapse (due to loss of pelvic floor support).
5. Complete tear may lead to incontinence to stool and flatus due to division of the sphincter ani muscle, after sometimes some patient will learn to contract the levator muscle and can control the passage of the solid faecal matter, but remain incontinence to liquid stools and flatus.
6. Residual recto vaginal fistula.
7. Dyspareunia from a tender scar in the vagina.

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3. DD of fecal incontinence.

1. Recto-vaginal fistula.
2. Old complete perineal tear.
3. Rectocele.
4. <u>Anal sphincter weakness:</u>
   a. Acquired: due to surgery as extension of median episiotomy/spinal cord trauma
   b. Congenital: as in case of spina bifida
5. Transient incontinence after prolonged labor.

1. *Recto-vaginal fistula:*

   Etiology:

   A. *Traumatic:*
   - Obstetric trauma: the commonest cause is badly healed complete perineal tear.
   - Surgical trauma: as injury to the rectum during posterior colpo-perineo-rraphy.
   - Other rare causes: defloration injuries and ulceration of an ill-fitting neglected pessary.

   B. *Inflammatory conditions:*

   As following rupture of a peri-rectal or peri-anal abscess.

   C. *Malignant R-V fistula:*

   Due to direct extension of malignant disease from the cervix, vagina or anterior rectal wall.

   D. *Post irradiation:*

   Usually associated with severe proctitis.

   E. *Congenital:*

   Very rare.

   Symptoms:
   - Large R-V fistula: loss of voluntary control over passage of feces and flatus and persistent leucorrhea due to secondary vaginal infection.
   - Small R-V fistula: involuntary escape of flatus which the patient feels as coming from the vagina.

2. *Old complete perineal tear:*

   Definition and etiology:

   An old complete perineal tear is a 3rd degree perineal laceration that has occurred usually during child birth but was missed at the time of delivery or improperly sutured and repaired.

   Clinical picture:

   See Q1

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4. Causes, diagnosis and ttt of recent and old complete perineal laceration.

1. *Recent perineal laceration:*

   Causes:

   1. Bad management of 2nd stage of labour (commonest cause):
   - Allowing premature extension of the fatal head before crowning.
   - Lack of adequate perineal support during delivery.
   - Instrumental delivery with inadequate episiotomy.

   2. Inadequately performed episiotomy in association with:
   - Delivery of malpositions and malpresentations associated with larger diameter of delivered head, as in case of direct occipto-posterior, face mento-anterior and after coming head in breech deliveries.
   - The use of instrumental delivery as forceps or vacuum extractor.
   - Rigid perineum (elderly multiparous, previous perineal scar).
   - Narrow subpubic angle → displacing head posteriorly with excessive perineal stretch.

   3. Rapid delivery of the head through the birth canal = precipitate labour.

   4. Severe oedema of the vulva with friable easily torn tissue (as in PE)

5. **Rarely, direct external trauma as with fall from a height, car accidents or defloration injuries.**

Diagnosis:

The anterior portion of the sphincter ani externus muscle is involved, the rectal wall may be torn leading to prolapse of the rectal mucosa

A. *Symptoms:*
   1. History of PPHge and peurperal sepsis
   2. Dyspareunia and sexual dissatisfaction

B. *Signs:*
   1. Prolapse
   2. Incontinence
   3. Fistula

Management:

1. *Prevention:*
   a. Proper management of 2nd stage of labour, by maintain flexion of fetal head until crowning occur, then allowing for slow delivery of the head in between uterine contraction.
   b. Episiotomy, when the perineum threatens to tear and routinely with instrumental delivery.
2. *Treatment:*
   - Every perineal tear, however small should be repaired.
   - 1ry suture is possible if done within first 24 hours.
   - If the case is seen later than that, it is considered as a septic wound and left to heal by granulation, repair in such cases is postponed until all signs of infection have disappeared, usually 3-6 months later.
   - Post-operative care after perineorrhaphy aims to keep wound dry and clean to encourage healing by 1ry intention.
   - <u>Recent 3rd degree perineal tear:</u>
     a. The rectal wall is sutured in 2 layers by delayed absorbable type of suture, first continuous then interrupted suture not going through the rectal mucosa, the suture should extend well above the apex of the laceration.
     b. The anal sphincter, the cut ends of the anal sphincter are identified and sutured.

2. *Old complete perineal tear:*

Causes:

A 3rd degree perineal laceration that has occurred usually during child birth but was missed at the time of delivery or improperly sutured and repaired.

Diagnosis:

Includes clinical picture (mentioned in Q1)

Treatment:

- Essentially surgical after pre-operative preparation.
- The patient is admitted 3 days before the operation, during which:
  a. A purge is given to empty the bowel with daily cleansing enema and vaginal douches.
  b. The patient is kept on a non-residue fluid diet (free of milk).
  c. Intestinal anti-septics (neomycin or streptomycin).

- Post operative care:

a. The vulva is regularly washed with antiseptic solutions then dried (at least 3 times daily).
b. The low residue diet is continued, as well as the intestinal antiseptic.
c. Antibiotics against wound infection (systemic and possibly local creams or gel).
d. 'The vaginal pack is removed after 24 hours.
e. On the fifth night the patient is given oral purgative solutions (as 50 ml. castor oil) in order to lubricate the stools. After that the patient is given daily oral laxatives and stool softeners to avoid constipation.

N.B.

In the event of subsequent pregnancy, a postero-lateral episiotomy should be done before delivery of the head to avoid recurrence of the laceration.

Mnemonic:

Post-operative care:

NPO لمدة يوم يومين
Low residue diet لمدة أسبوع أسبوعين
ومفيش **suppositories** لمدة شهر شهرين
اليوم الخامس تديها ملين
و تكمل معاها **oral neomycin**
و**antibiotics** أخرى **systemic** و **local**
و الله يكرمك متنساش ال**care** ال**local**
و مفيش **intercourse** لمدة شهرين
و الولادة الجاية اعمل **episiotomy** كبيرة على جنب من الاتنين

1. **On the fifth day, 50 ml castor oil is given.**
2. **Neomycin is given before OP and continued after as intestinal antiseptic**
3. **Systemic antibiotics should contain metronidazole**
4. **Local care "DRY CLEAN PERINEUM"**
5. **Next delivery a posterolateral episiotomy should be performed.**

5. Treatment of rectovaginal fistula.

Treatment:

The treatment of non malignant Rv-fistulae is **essentially surgical.** Preoperative and postoperative care the patient are for the success of the procedure, and they generally follow same rules as that de- scribed for old complete perineal tears.

1. Fistulas in the **lower third** of the vagina:

   Managed by cutting the remaining bridge of tissue below the fistula, thus converting the fistula into a complete perineal tear (**Lawson Tait's operation**). The tear is now sutured in layers, in the same manner and order described under repair of complete tears.
2. Fistulas in the **middle third** of vagina:
   - May be closed in the same manner for dealing with **vesico-vaginal fistulas.**
   - An alternative procedure is to start the operation as in perineorrhaphy for rectocele and to extend the dissection of the recto-vaginal septum upwards above the fistula. The hole in the rectum is then closed, and the operation continued as a perineorrhaphy.
3. Fistulas in the **upper third:**

   High recto-vaginal fistulas are usually surrounded by dense fibrosis, and are difficult to close vaginally. They are usually best dealt with by an **abdominal (trans-peritoneal operation) operation.**

Urinary incontinence & fistula

1. Define different types of urinary incontinence. (June 2014)

Urinary incontinence:

The involuntary leakage of urine that is objectively demonstrable.

Types:

1. *Urethral:*
    a. Stress urinary incontinence
    b. Urge incontinence
    c. Retention with overflow
    d. Nocturnal enuresis
    e. Urethro-vaginal fistula
2. *Extra-urethral:*

    Genitourinary fistulas apart from urethro-vaginal type

Stress urinary incontinence:

- The involuntary escape of urine through the urethra during suddenly increased intra-abdominal pressure as during cough, sneeze, or laughing.
- Urodynamic stress incontinence: is term used when stress urinary incontinence is confirmed with objective urodynamics testing.

Urge incontinence:

Involuntary leakage of urine through the urethra before starting to void when the bladder is partially full, posing difficulty in postponing urination.

Genito-urinary fistulas:

Are abnormal communication between the urinary organs (ureter, bladder, urethra) and genital organs (vagina, cervix, uterus). They are usually associated with continuous leakage of urine through vagina.

Retention with overflow:

Involuntary release of urine from an overfull urinary bladder, with absence of desire to void urine.

Nocturnal enuresis:

Involuntary urination at night while sleeping.

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2. Genuine stress incontinence; mention: definition, pathophysiology and treatment. (June 2010)

Definition:

- The involuntary escape of urine through the urethra during suddenly increased intra-abdominal pressure as during cough, sneeze, or laughing.
- Urodynamic stress incontinence: is term used when stress urinary incontinence is confirmed with objective urodynamics testing.

Pathophysiology:

- The bladder neck and proximal urethra are normally situated in an intra-abdominal retropubic position resting on the pelvic floor muscles and supported by the pubo-urethral ligaments.
- The position allows equal transmission of IAPR to the bladder and the proximal urethra,

maintaining a persistently higher IUPR over the IVPR.

- The difference pressure gradient results in urethral closure and continence even with abrupt increase in the IAPR, except during voiding.
- Descent of bladder neck and proximal urethra below symphysis pubis due to damage to levator ani muscles or the pubo-urethral ligaments, will make them no longer intra-abdominal organs and will results in unequal transmission of IAPR to the bladder and urethra.
- During sudden increase in IAPR the IVPR will exceed IUPR and urine will involuntary escape through the urethra leading to SUI, which is limited to the period of increased IAPR as during cough, sneeze, or laughing.
- The patient neither has the desire to void nor the control of voiding.

Treatment:

1. *Conservative measures for mild cases:*
    a. Pelvic floor muscles strengthening exercise:
        - Active pelvic floor muscle training (Kegel exercises).
        - Passive electrical pelvic floor muscle stimulation.
    b. Estrogen therapy:
    Vaginal cream in cases of menopausal atrophy may improve urethral blood flow and alpha-adrenergic receptor sensitivity.
    c. Pessary treatment:
    In some cases of vaginal prolapse may improve associated SUI.
2. *Surgical treatment for moderate and severe cases:*
    a. Bruch colpo-suspention operation:
        - It is abdominal procedure in which (upper vagina at level urethrovaginal junction and upper third of the urethra) is plicated on each side of ilio-pectineal ligament.
        - This operation is considered gold standard procedure with success rate of 95% in 1 year and nearly 75% after 15 years.
    b. The sling procedures:
        i. Tension free vaginal tape (TVT):
            - TVT is performed using synthetic mesh like tape introduced vaginally on each side of the proximal urethra, passing just below it acting as a sling then each end of the tape is pushed upwards to be pulled through abdominal wall.
            - TVT has high success rate up to 90% with advantage of simple technique that require short stay in hospital con be performed under local anesthesia.
        ii. Trans obturator tape (TOT):
            - TOT is performed using synthetic mesh like tape introduced vaginally passing from one obturator Forman to other piercing obturator diaphragm and forming a sling below the proximal urethra, and distal end pulled out through small groin incision at the level of clitoris.
            - TOT is similar to TVT with less complication performed with regional anesthesia
    c. Kelly's peri urethral fascial plication sutures:
        - It is vaginal procedure in which peri urethral plication of fascia is performed along the course of a cystourethrocele repair.
        - It is associated with success rate up to 70% but unfortunately will fall to only 30 %

after 5 years.

d. Peri-urethral injection of collagen.

---

3. Investigation of stress urinary incontinence. (June 2013)

1. *Urodynamics.*
2. *Urine analysis to exclude UTI*
3. *Preoperative investigations for ppt factors:*
   a. *Labs:*
      CBC, blood sugar, urine analysis
   b. *Radio:*
      - Chest x-ray
      - US to exclude any associated pathology

      Urodynamic studies include: Cystometry, uroflowmetry & urethral pressure profile.

1. *Cystometry:*
   a. Involuntary leakage of urine through the urethra during increased IAPR.
   b. Absence of detrusor contraction during filling cystometry.
2. *Urethral pressure profile:*
   a. Increase IVPR > IUPR during straining.
   b. Decreased functional urethral length (length oh urethra closed by IUPR).

---

4. Stress incontinence etiology, diagnosis and prevention. (june 2012)

Etiology:

'As prolapse without iatrogenic part"

1. Weakness of pelvic floor muscle and support:
   a. **Congenital**: occurring in young and nulliparous women with no risk factors.
   b. **Childbirth trauma**: due to overstretching of pelvic floor muscle and the endopelvic fascia with damage of its nerve supply especially after prolonged and difficult deliveries.
   c. **Postmenopausal**: due atrophic changes affecting pelvic fascia **2ry to estrogen deficiency.**
2. Anterior vaginal wall prolapses:
   Due to descent of bladder neck and proximal urethra.
3. Chronic increase in IAPR:
   Marked obesity, chronic lung disease or chronic constipation may precipitate condition in women with weak pelvic floor musculature.

[A sidenote: Smoking cause SUI by 2 ways:

1-Anti-estrogenic effect → ↓collagen synthesis → Atrophy of the urethral mucosal seal (↓ uretheral mucosal coaptation ,an important factor in maintaing urinar continence) →↓urethral resting pressure

2-Persistent ↑ I.A.P due to chronic cough]

Diagnosis:

1. *Clinical picture:*
    a. Symptoms:
        - Involuntary leakage of urine through the urethra during cough, sneeze, or laughing.
        - Symptoms of associated prolapse as heaviness and mass filling vagina may present.
    b. Signs:
        - Cough stress test: electing involuntary escape of urine through the urethra during cough while bladder is partially falling ang patient in erect or lithotomy position.
        - **Bonney's test:**
            - With the patient lithotomy position perform a cough test to elicit SUI.
            - The bladder neck is then elevated gently by index and the middle fingers of the examiner hand placed in vagina on either side of the urethra without compressing it.
            - The patient is asked to cough:
                - If no urine escape: then the bladder neck descent is the cause.
                - If urine escape: then the weakness of the bladder neck will be the cause.
        - **The Q-tip test:**

            To detect mobility of urethra-vaginal junction on straining. More than 30-degree mobility indicates hypermobile urethra-vaginal junction.
        - **Presence of genital prolapse should be excluded** "discuss".
2. *Investigations:*

    As previous Q

Prevention:

1. Avoid prolonged 2nd stage of labour and minimize child birth trauma.
2. Pelvic floor exercises especially in the puerperium or after pelvic surgery.
3. Avoid marked obesity and overweight.
4. Treatment of chronic cough and constipation especially in the post-menopausal period.

---

5. Stress urinary incontinence; definition, etiology, pathophysiology and clinical picture. (Jun 2015)

As mentioned above.

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6. Compare stress and urge incontinence as regard: definition, diagnosis, treatment. (sep 2014)

Point of comparison	*Stress urinary incontinence (SUI)*	*Urge urinary incontinence*
Definition	The involuntary escape of urine through the urethra during **suddenly increased intra-abdominal pressure** as during cough, sneeze, or laughing.	Involuntary leakage of urine through the urethra **before starting to void** when the bladder is partially full, posing difficulty in postponing urination.
Diagnosis		
<u>Symptoms</u>		
Desire to void	Absent	Present (sudden)
Nocturia	Absent	May be present
Associated genital prolapse	Present	Absent
<u>Investigations</u> (Cystometry & urethral pressure profile)		
Detrusor overactivity (DO)	Absent	Present
Increased IVPR>IUPR during straining	Present	Absent
Decreased functional urethral length	Present	Absent
Treatment	1- Pelvic floor exercise 2- Scheduled voiding 3- Estrogen therapy 4- Pessary treatment **<u>Surgical</u>** 1- Kelly's Periurethral fascial plication sutures 2- Burch colposuspension 3- Sling procedures (TVT-TOT)	**<u>Medical</u>** Anticholinergic drugs (detrusitol 2 mg x 2 times per day) + Bladder training exercises

---

7. Etiology and symptoms of vesico-vaginal fistula. (june 2016, 1998)

Etiology:

1. *Congenital:*
   During division of urogenital sinus → rare
2. *Traumatic:*
   a. **<u>Obstetric:</u>**
      - Direct: forceps

- Indirect: CPD and obstructed labor

b. <u>Surgical:</u>

esp. if there are adhesions

c. <u>Direct:</u>

Foreign body or fracture pelvis

3. *Neoplastic:*

   e.g. cancer cervix

4. *Inflammatory:*

   Rupture of abscess

5. *Irradiation.*

Symptoms:

1. **<u>Complete urinary incontinence:</u>**
   - Continuous leakage of urine through vagina in patient with history of recent delivery, pelvic surgery or pelvic irradiation is the main presenting symptom.
   - Loss of desire to void urine due to absence of bladder filling is the rule especially when the fistula is large and in allow position.
   - In **necrotic fistula**; incontinence may present **days or weeks** after a **difficult delivery** or months after **pelvic irradiation.**
   - In **traumatic fistula**; leakage of urine starts almost **immediately.**
2. **<u>Partial urinary incontinence:</u>**

   Interrupted leakage of urine with occasional desire to micturate may occur in some cases with **small** VVF situated **high** in the bladder enabling the patient to retain some of urine in the bladder.
3. **<u>Pruritus and vulvitis:</u>**

   These may occur secondary to prolonged irritation of vulva and vaginal skin with leaking urine.
4. **<u>Cystitis and UTI:</u>**

   May complicate prolonged or neglected cases due to ascending infection from vulva.

---

8. Causes and diagnosis of uretero-vaginal fistula. (june 2004)

Causes:

Occurs as a result of injury to the ureter during gynecological operations as hysterectomy but may also develop following a difficult delivery by CS.

Diagnosis:

A. Symptoms:

Incomplete urinary incontinence; the bladder fills normally from the intact ureter and the patient voids normally with a preserved sensation of the bladder filling and desire to micturate while the urine continuously dribbles through the vagina from the affected ureter at the site of UVF.

B. Inspection by sim's speculum:

The fistula is always small situated high in the vagina lateral to the cervix.

C. Investigations:

- UVF con be differentiated from VVF by methylene blue test.
- Cystoscopy shows a normal bladder with presence of ureteric efflux only on one side.

[A side note: Both Unilateral UVF & small high valvular VVF have the same symptoms i.e partial urinary incontinence
while low &/or large VVF has the same symptoms as bilateral UVF]

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9. Diagnosis and management of vesico-vaginal fistula.

Diagnosis:

A. History (etiology):

See before.

B. Symptoms:

See before.

C. Signs :

a. <u>With the patient in Sim`s position :</u>

Inspection of the anterior vaginal wall is performed using sim`s vaginal speculum , to visualize fistulous opening & detect urine leakage from VVF orifice .

b. <u>With the patient in lithotomy position :</u>

Digital palpation of the anterior vaginal wall is performed to detect the site , size , of VVF and surrounding scarring & fibrosis.

D. Investigations :

a. <u>Retrograde coloured dye injection into the bladder.</u>

Retrograde injection of 200 ml. of coloured dye (methylene blue or indigo carmine) into the bladder is performed via a urethral catheter. Coloured dye will be seen dribbling from the fistulous opening at the anterior vaginal wall using sim`s speculum .

b. <u>Intravenous pyelography (IVP) .</u>

It is important to:

- Evaluate the amount of residual urine in the bladder.
- Delineate course of ureters.
- Exclude ureteric fistulae.

c. <u>Cystoscopy & urethroscopy.</u>

i. Cysto-urethroscopy:

Helps to exclude urethral fistulae & delineate the fistulous track to the vagina.

ii. Cystoscopy:

- Visualizes VVF & determines their relation to the ureteric openings in the bladder.
- Exclude multiple VVF & ureteric fistulae.
- Can reveal any associated bladder pathology.

iii. Urethroscopy:

May exclude associated urethrovaginal fistulae.

Management:

A. Prevention :

- Avoid prolonged & obstructed labour by prompt diagnosis of CPD .
- Use of CS instead of difficult forceps procedures .
- Proper surgical technique in pelvic surgery to avoid bladder or ureteric injury .

- Immediate intraoperative repair of bladder & ureteric injury once diagnosed .

B. Conservative management

- In case of a necrotic fistula , detected few days after a difficult labour , an **indwelling silicone catheter** is introduced through the urethra and left for a period of 4-6 weeks , to divert the flow of urine away from the fistula , prevent bladder distension & allow wound healing .
- Surgical repair is usually postponed for 3-6 months until edema subsides and tissue involution is complete, where surgery becomes easier & safer allowing for better dissection & healing.
- Spontaneous closure of a small VVF may be achieved in some cases with complete healing of the tear, or failing this, it will be left much smaller in size.

C. Surgical treatment :

- Surgical anatomical repair is **the gold standard** in management of VVF .
- The **1st attempt** for fistula repair carries the **best prognosis**. Repeated trials for repair are less successful due to extensive fibrosis left after each attempt
- Proper postoperative bladder drainage is crucial to success of surgery.

D. Preoperative preparation:

- Confirm site, size & number of the fistulas & exclude associated UVF by cystoscopy & IVP
- Protection of vulva & vagina from continuous irritation by dribbling urine via indwelling catheters, vaseline or zinc oxide ointments
- **Culture & sensitivity of urine**, if a pathogenic organism is found → Give antibiotic until urine is sterile.

E. Operations :

- *Vaginal:*
  - Dedoublement
  - Sim's saucerization
  - Latzko
- *Abdominal :*

  If high, recurrent or ureteric

F. Postoperative care:

1. The bladder should be kept constantly empty to avoid any tension on the sutures via ensuring patent catheter
2. Catheter never removed before 10-14 days (after that micturition should be every 2 h in the morning & every 4 h at night)
3. In case of successful repair of VVF pregnancy should be avoided for **1 year after the operation** & at full term **delivery by CS is mandatory** to avoid recurrence & other complications of vaginal delivery on the pelvic floor.

6.INFECTIONS & BENIGN CONDITIONS OF VULVA AND VAGINA

Infections

1. **Post menopausal defense mechanism "natural barrier" against vaginal infections. Enumerate common types of infections in the reproductive age. (June 2010)**

Natural defense mechanisms in menopausal woman:

Only cellular and humoral immunity (not vaginal epithelium or vaginal pH).

Common types of infections in the reproductive age:

1. Bacterial vaginosis " non-specific vaginitis ""gardnerella vaginitis"→ commonest
2. Candida vaginitis " candiasis""monaliasis"→ 2nd most common
3. Trichomonas vaginalis → 3rd most common

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2. **Bacterial vaginosis: etiology,clinical picture (June2005/2009)**

Bacterial vaginosis is **the commonest bacterial vaginal infection** encountered in the gynecologic practice.

Etiology:

- Bacterial vaginosis represents a state of unexplained alteration of the normal vaginal flora where:
  1. Anerobic organisms "Gardnerella vaginalis-bacteriodes species & mycoplasma hominis" markedly increase in concentration "thousands of times"
  2. Hydrogen peroxide producing lactobacilli markedly decrease "or even disappear"
  3. The vaginal PH rises, ranging 4.7-7.0 "alkaline PH"
- Anerobic bacteria produce enzymes that breaks peptides to amino acids & amines resulting in its characteristic discharge & odour
- It is not STD hence the sexual partner does not need to be treated

Clinical picture:

1. *Symptoms*
   a. Asymptomatic in 50% of women
   b. Vaginal discharge is the commonest symptom. It is usually profuse, thin, homogenous, non irritant, malodorous discharge
   c. Fishy amine characteristic smell is especially noticed around time of mensisor following sexual intercourse
2. *Signs*

   Thin, homogenous discharge adherent to vaginal walls "giving milk spilled over tissue appearance"

---

3. Candida vaginitis: causative organisms –clinical picture –ttt (sep 2001/2012- June 2014)

Causative organisms:

1. Candida albicans "fungus causing yeast infection" in >80% of cases.
   It is a normal inhabitant of the bowel & peri-anal region in almost 30% of women with no symptoms of infection
2. Candida tropicalis & Torulopsis glabrata less commonly encountered in about <20% of cases. They are usually resistant to standard antifungal treatment regimens.

Clinical picture:

Symptoms:

- Intense pruritus with itching with progressive scratching "main symptom"
- Vaginal burning sensation that may cause discomfort & dyspareunia
- Vaginal discharge : scanty thick white discharge "cottage –cheese discharge"
- Dysuria may be an associated symptom in some cases

Signs:

- Vulva may be red & swollen – sometimes showing classic satellite lesions
- Vagina may show patches of scanty adherent cottage-cheese discharge

Treatment:

1. *Oral preparations :*

   Fluconazole single dose 150mg → un complicated cases
2. *Intra vaginal preparations:*
   - **Butoconazole** 2% 5g daily cream for 3 days Or
   - Clotrimazole 2% 5g daily cream for 3days Or
   - **Miconazole** 4% 5g daily cream for 3 days Or
   - Ticonazole 6.5% 5g ointment → single application Or
   - Terconazole 80mg vaginal suppository –one sup. For 3 days
   - Common side effect: hepatotoxicity.
   - During pregnancy : intravaginal ttt for CV → safe throughout pregnancy
   - Cure rates of >80-90% expected within one week of ttt
   - Routine ttt of sexual partners → not recommended except in recurrent infection

---

4. Discuss management of recurrent monilial vaginitis (sep 2016)

Diagnostic C/P:

See previous question.

Diagnostic investigations:

1. Search for predisposing factor: **DIABETES?** Immunocompromised? COCs? Corticosteroid therapy?
2. Check husband
3. Check if non-albicans by culture

Treatment:

1. Treat predisposing factor (DM, discontinue COCs and CS and husband)
2. Give prolonged antifungal therapy "oral and vaginal" and monitor liver functions as antifungals are hepatotoxic

---

5. Give the incidence of T. vaginalis – describe the organism, mode of infection and ttt "Aug 2009"

Incidence:

It is the **3rd common cause** of vaginitis accounting for almost 25% of cases presenting with vulvo-vaginitis with vaginal discharge

Organism:

T. vaginalis is an ovoid –motile – flagellated protozoon with 4 anterior flagellae & an axostyle which traverses its body to end in a spike. It is slightly larger than leucocyte "20mm length & 10mm width"

Mode of infection:

In 70% of cases, TV can be recovered from male partners. So, it is considered a sexually transmitted disease (STD)

Treatment:

- -Metronidazole 2g (orally) → single dose Or
- Tinidazole 2g (orally) → single dose
- During Pregnancy: Oral Metronidazole after 1st trimester
- Male partner → should be prompthy treated by oral metronidazole
- Recurrent & Resistant : if re-infection is excluded → patient can be treated with metronidazole 500mg twice daily /7days
  [Remember: like BV]

---

6. Trichomonas Vaginitis "June 2000-2001/Sep 2013"

Incidence, etiology & ttt:

See before.

Diagnostic C/P:

- Asymptomatic in 25% -50% of women
- Vulvar irritation → vulvitis & pruritis
- Vaginal discharge: Copious yellow or green frothy & offensive "fishy odour" discharge
- Speculum exam → reveals vaginal or cervical punctate hemorrhage in 25% of cases known as (**Strawberry Spots**)

Diagnostic investigations:

- Vaginal PH → weak acidic usually "5-6"
- Saline wet preparations of vaginal discharge → numerous leucocyte of highly motile flagellated trichomonas in 70% of cases
- Cultures are sensitive but not widely used (they need special media which isn't available in man labs)
- PAP Smear → while screening for cervical neoplasia trichominiasis may be noted with a sensitivity approaching 60%

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7. Cervical erosion (June 2002/2004)
Causes, diagnosis & ttt (June 2016)

Definition:

Cervical erosion (ectopy) is a bright red area around the external OS due to replacement of the ectocervical stratified squamous ep. with the endocervical columnar ep., which is thin & showing underlying vessels.

Aetiology:

1. *Chronic cervicitis:*
   - The infected cervical discharge from endo-cervix → devitalized denuded area of the stratified squamous epith. around the ext. os.
   - 1st healing phase: columnar epithlium from the endo cervix grow to cover the denuded area of ecto cervix
   - 2nd healing phase: st. sq. epith. of ectocervix grow under col. epith. resulting in blockage of ducts of cervical glands &formation of retention (Nabothian)cysts
   - Exacerbation of infection leads to repetition of the whole process
2. *Hormonal erosion:*

   Excess estrogen "during pregnancy or with COC use" causes growth of col. epith. replacing st. sq. epith.
3. *Congenital erosion:*

   Persistence of intrauterine condition where col.epith. covers an area of the ecto cervix " very rarely seen after puberty"

C/P:

Symptoms:

- Vaginal discharge: excessive, mucoid in nature
- Contact bleeding: manifested after intercourse or douching
- S/O of chronic cervicitis if complicated by infection associated → mucopurulent
- Foul discharge, backache, dysmenorrhea, dyspareunia, contact bleeding , frequency of micturition & infertility

Signs:

- Vaginal examination: velvety sensation and occasional contact bleeding
- Speculum examination: reveals flat erosion (red flat area) or papillary
- Erosion (red area with raised folds) or follicular erosion (red area is glandular distension) [+ signs of chronic cervicitis: Nabothian cysts, chronic hyper trophic cervicitis]

Investigations:

Vaginal, cervical smears to exclude malignancy

Treatment:

1. Hormonal erosion: no ttt, except if persists for more than 3 months.
2. Antimicrobials to treat associated infection as chronic cervicitis, mainly chlamydia & N gonorrhoea)
   - Azithromycin 1 gm orally single dose Or
   - Doxycycline 100 mg orally twice daily/7days
3. Follow up should be done as recurrence & re infection isn't rare.
4. Resistant or recurrent infection are mainly treated by repeated antibiotic courses
5. Destruction of cervical tissue by cauterization, cryotherapy, or LASER (not commonly used)

---

8. Cervicitis (June 2006)

There is acute & chronic cervicitis.

1. Acute endocervicitis:

Definition:

It is acute inflammatory if endocervical glands, underlying tissue mostly 2 ry to infection by sexually transmitted organisms

Aetiology & causative organisms:

- Sexually transmitted organisms: chlamydia trachomatis , Neisseria gonorrhea &trichomonas vaginalis
- Also staph aureus& streptococci in relation to IUD insertion, purpureal , post operative or post-operative infection (D&C) procedures

C/P:

- S/O:
  Mucopurulent vaginal discharge, backache, dyspareunia & sometimes mild fever
- Signs:
  - On speculum examination : cervix is red swollen &mucopurulent discharge
  - On bimanual :marked tenderness on moving the cervix

Diagnosis:

Culture & sensitivity of the discharge: (Thayer-Martin for gonorrhea), (Mac Coy cells for chlamydia) for appropriate antibiotic therapy

Treatment:

- Azithromycin 1 gm orally single dose
- Doxycycline 100 mg orally twice daily /7 days
- Concurrent Gonococcal infection should be considered as prevalence of gonorrhea is high among patient population under assessment
- Un complicated genital , urinary & rectal infection:
  Ceftriaxone 250 mg IM single dose + above mentioned ttt
- Sexual partner should be treated as well

Complications:

1. Chronic cervicitis due to the racemose (branching) nature of the glands & lack of cyclic shedding of cervical mucosa
2. Spread of the infection to upper genital tract may lead to PID.

2. Chronic cervicitis :

Definition:

Chronic inflammation of endo cervical glands , it is a common sequel of acute cervicitis

C/P:

- *S/O:*
  - Mucopurulent offensive vaginal discharge in association with endocervicitis
  - Backache (dt spread of infection along the uterosacral ligament)
  - Dyspareunia (due to parametrial if present)
  - Dysmenorrhea (due to pelvic congestion)
  - Contact bleeding (associated with congestion and cervical erosions)
  - Infertility (dt infected hostile cervical discharge)
  - Frequency of micturition(dt. Cystitis caused by lymphatic spread)

- *Signs (on speculum examination):*
  - Mucopurulent discharge coming out from the cervix
  - Cervical erosion
  - Chronic hypertrophic cervicitis (swollen &hyper emic cervix)
  - Mucous polyp (reddish pedunculated small polyp protruding from the endo cervical canal due to hyperplasia of endo-cervical columnar epithelium
  - Nabothian cyst: small blue or yellowish cyst within the substance of cervix and projection on the portio-vaginalis. It represent distended cervical glands e secretions due to blockage of their ducts

Diagnosis:

- C & S of the discharge
- Exclusion of malignancy by cervical & vaginal smear in cases of suspicions cervix

Treatment:

1. Oral or vaginal antimicrobials according to C&S
2. Resistant or recurrent infections are mainly treated by repeated antibiotic courses
3. Destruction of cervical tissue by cauterization, cryotherapy or LASER (not commonly used)

---

9. Chlamydia trachomatis infection of cervix: clinical picture and ttt (june 2015).

Clinical picture:

A. *Symptoms :*
   - Almost asymptomatic in 75% of cases
   - Symptomatic cases: mucopurulent endocervicitis (mucopurulent discharge with edematous and hyperemic cervix)
   - Symptoms of urethritis & pyruia: in association with negative urine culture is highly suggestive in sexually active women
   - Fever & lower abdominal pain suggest PID which occur in 40% of women with untreated chlamydia. (It is more gradual & protracted than gonococcal PID)

B. *Signs:*

   On speculum examination , cervix is red swollen and mucopurulent discharge.

Treatment:

- Azithromycin 1 g orally once 4 tablets 250 mg each (safe in pregnancy)
- Or doxycycline 10 mg orally twice/daily /7 days (not used in pregnancy)
- Abstinence from sexual intercourse for 7 days from start of ttt
- Test of cure is not recommended

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10. Diagnosis of acute salpingitis (1999)

Diagnostic symptoms & history:

A. *History:*

   Recent IUD insertion, D& C procedure, recent delivery or abortion , recent performed HSG for infertility, in young sexually active woman

B. *Symptoms:*
   - Often start in the post menstrual period.
   - Women with chlamydia PID usually have clinically milder diseases than those with

gonococcal PID. Generally nonspecific; acute lower abdominal pain (most common symptom)

- Fever, headache, malaise
- GIT s/o as nausea & vomiting
- + Vaginal discharge & sometimes vaginal bleeding

Diagnostic investigations:

A. *Examination of cervico-vaginal discharge:*
   - Gram stain: useful in diagnosis if gonorrhea although alone may miss up to ½ of cases & doesn't diagnose chlamydia trachomatis
   - Testing for chlamydia & gonorrhea antibodies will help diagnosis in cases where clinical s/o are suggestive of acute PID

B. *Blood tests:*
   - Increases WBCs (leukocytosis) is not accurate for diagnosing acute PID
   - + ESR is nonspecific finding although elevated in > 75% of PID cases (good sensitivity but low specificity)

C. *Ultra sound:*
   - Finding may be normal or non-specific
   - TVS to exclude ectopic pregnancy, uterine myomas & ovarian swellings

D. *Laparoscopy:*
   - The gold standard to confirm the diagnosis whenever in doubt or in cases with poor response to parenteral antibiotic therapy after 48-72 hours of initiating TTT
   - Laparoscopy will reveal: tubal serosal hyperemia, tubal wall edema & purulent exudates from the fallopian tubes pooling in the cul-de-sac

E. *Culdocentesis :*

   Aspiration pf purulent fluid in the pouch of douglas may assist diagnosis & antibiotic selection after culture & sensitivity, yet its use is limited by its associated pain, & tendency for secondary infection

F. *Testing for HIV & STDs:*

   Should be offered to all women diagnosed as PID

CDC diagnostic criteria (2010):

A. *Minimum diagnostic criteria:*
   - Sexually active young female with any risk factor for STDs
   - ± Lower abdominal pain
   - ± Uterine or adnexal tenderness on bimanual pelvic examination
   - **Or** cervical motion tenderness on bimanual pelvic examination

B. *Additional criteria:*
   - Oral temperature > 38.3 C
   - Mucopurulent vaginal/cervical discharge
   - Numerous WBCs on wet mount preparation
   - Increased ESR &/or CRP
   - +ve endocervical tests for cervical N. gonorrhea &/or trachomatis)

C. *Most specific criteria:*
   - Endometrial biopsy showing histopathological evidence of endometritis
   - Tubo-ovarian complex seen on pelvic US or MRI

- Laparoscopic evidence of PID

D.D of acute PID = DD of acute abdomen:

1. Disturbed ectopic pregnancy
2. Acute salpingitis
3. Torsion, rupture or hemorrhage in an ovarian cyst
4. Red degeneration in a fibroid or torsion of pedunculated subserous fibroid
5. Hemorrhage in corpus luteum

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11. Clinical picture of TB of the female genital tract (june 2013)

- T.B is a chronic granulamatous disease caused by mycobacreium tuberculosis.
- Female genital TB is always secondary to a lesion elsewhere in the body, most commonly the lungs. Spread of infection is mainly hematogenous

Clinical picture:

A. Symptoms:

- Early cases are usually asymptomatic
- More advanced disease will usually present with inferitity associated with symtoms& signs suggestive of PID with or without menstrual disorder
    1. Infertility: genital TB may be the underlying cause in almost 5-10% of infertile women mostly due to : tubal destruction , peritoneal adhesions or endometrial synechiae
    2. Chronic pelvic pain associated with low grade fever , is a common presentation.
    3. Acute lower abdominal pain with high fever may accuse if 2ry infection develops on top of a TB pyosalpinx
    4. Abdominal bleeding may accuse from ulcerative lesion in the endometrium
    5. 2ry amenorrhea or oligomenorrhea may occur in about 10% of cases due to interuterinesynachiae , and/ or ovarian destruction

B. Signs:

- Most cases are normal on gynaecologic examination.
- A tender, fixed adnexal swelling, with low grade fever especially in an infertile patient with no previous history of pelvic infection strongly suspicious for TB
- Genital ulcerative lesions :with serpiginous margins & underminded edges, characteristic of TB , my develop on the vulva & cervix , with later scarring or sinus formation

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12. Gonorrhea infection: organism, clinical picture, complication & ttt (sep 2014)

Gonorrhea is a sexually transmitted disease.

Organism:

- Caused by gram –ve diplococcus Neisseria gonorrhea, a bacterium species that involves infection of columnar & transitional epithelium primarily causing gonococccal endocervicitis.
- The vagina is not involved being lined by squamous epithelium (except in childhood)

Clinical picture:

1. Cervicitis
2. PID

3. Urethritis
4. Bartholinitis
5. Disseminated

"اكتب اللي تفتكره جمبها .. مش لازم اللي في الكتاب"

For details:

- Frequently asymptomatic
- In symptomatic cases, symptoms usually appears 2-5 days after exposure, but may be delayed for up to 30 days. They include:
    1. Mucopurulent cervical discharge, as occurs in acute cervicitis
    2. lower abdominal pain, anemia & fever if PID develops
    3. Dysuria may occur in both men & women if there is associated urethritis 7 cystitis
    4. S/O of bartholinitis may occur if infection of Bartholin gland occurs
    5. S/O of pharyngitis & proctitis may be present & should be looked for
    6. Disseminated gonococcal infection (< 3% of cases), may cause arthritis, endocarditis & septicemia
    7. Other sequelae, include infertility & chronic pelvic pain.

Complications:

1. Female: PID, PTL, PROM, P. sepsis
2. Infant: eye, sepsis
3. Male: urethritis, prostatitis, infertility

Treatment:

CDC regimens for ttt of uncomplicated N gonorrhea (2010):

A. *Uncomplicated genital , urinary & rectal infection:*
    - Ceftraixome 250 mg IM single dose
      And
    - Azithroxycin 1g orally single dose
      Or
    - Doxycycline 100 mg a day for 7 days
- No test of cure is required
- Sex partners within 60 days of appearance of symptoms should be evaluated & healed for gonorrhea & chlamydia

B. *Disseminated gonococcal infection:*
    - Ceftriaxone 1g IM or IV every 24 hrs
    - Then Cefixime 400 mg orally twice daily to complete 7-12 days of ttt

C. *Treatment of PID:*
    Discuss.

13. Chlamydia trachomatis infection: sites, clinical picture, ttt & complications (june 2012/sep2011)

Sites:

It attacks only columnar ep.:

- Primary sites for infection: include the endocervix & less commonly the urethra
- Secondary sites for infection: upper genital infection (PID)

Clinical picture & ttt:

See before.

Complications:

1. Ascending infection may lead to chlamydial salpingitis & tubal damage that may predispose to ectopic pregnancy & infertility.
2. During labour of the vaginally delivered remote → chlamydial conjunctivitis may develop in 50% of cases & late onset of pneumonitis in 10% premature delivery & post-partum endometriosis are also associated patterns.

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14. Herpes simplex virus infection (sep 2011)

Herpes simplex virus is a double strand DNA virus of 2 main types:

1. *HSV-1:*
   - Primarily transmitted by non –venereal routes particularly following contact with infected saliva.
   - Responsible for facial & oropharyngeal infection like herpetic gingivitis –stomatitis & the common cold sore or fever blister .
2. *HSV-2:*
   - Usually transmitted by venereal route to the sexual partner or maternally to the new born during vaginal delivery.
   - Responsible for genital herpes & neonatal infection , has been linked epidemiologically with carcinoma of the cervix

C/P:

- The 1st attack is the most serious with pain, vulvitis, lymphadenopathy & discharge. It occurs after an incubation period of around 21 days, and is manifested by multiple painful ano-genital vesicles or painful ulcers with erythematous case that usually resolve after 3-4 weeks
- Subsequent attacks: are normally shorter & less severe

Diagnostic investigations:

1. Culture of serum collected from vesicles by aspiration or by swabbing base of the ulcer
2. Serum anti HSV antibody levels are increased with both types type 2 & type 2 infection

Treatment:

1. Mainly supportive with analgesics
2. TTT of any associated 2ry infection
3. 1st attack: acyclovir 400 mg orally three times a day/ 7-10 days
   TTT can be extended if healing is incomplete
4. Subsequent attacks: acyclovir 400 mg three times a day for 5days

---

Benign conditions of vulva and vagina

Vulvar ulcers

1. Give an example for each of the following and its line of treatment (june 2009).

A. Painless ulcer of the vulva:

1. *Syphilitic ulcer:*

   It is a primary syphilitic lesion treated by:

   - Benzathine penicillin G 2.4 million units IM single dose.
   - Doxycycline 100 mg orally twice daily for 2 weeks in non pregnant patients with penicillin allergy.

2. *Granuloma inguinale:*

   Treated by:

   - Doxycycline 100 g orally twice a day for at least 3 weeks & until all lesions have completely healed
   - OR Azithromycin 1 g orally once per week for at least 3 weeks & until all lesions have completely healed

B. Very painful ulcer of the vulva

Herpetic ulcers:

Treated by:

- Mainly supportive with analgesics.
- Treatment of any associated secondary infection.
- First attack Acyclovir 400 mg orally three times a day for7-10 days and can be extended if healing is incomplete.
- Subsequent attacks: Acyclovir 400 mg orally three times a day for 5 days.

Non-neoplastic swellings of vulva

2. List; non neoplastic swellings of vulva and write a short essay on the commonest one (june2012)

Non neoplastic swellings: (Congenital-Traumatic-Inflammatory-Miscellaneous)

1. *Congenital hypertrophy of the clitoris.*
2. *Retention cysts:*
   a. Bartholin duct cysts
   b. Sebaceous cyst
   c. Epidermal inclusion cyst (May be traumatic following episiotomy or circumcision or may be congenital)
   d. Hydrocele of the canal of Nuck
3. *Endometrioma*
4. *Traumatic vulvar hematoma*
5. *Circulatory disorders (Varicose veins or edema)*
6. *Inflammatory conditions (specific or non-specific)*

Bartholin's duct cyst

- The **commonest vulvar swelling**. It is actually **a cyst of the duct and not of the gland.**
- It contains mucoid fluid and is lined by transitional epithelium.
- **Anatomy:**
  - They are bilateral compound racemes glands situated deep in the labia majora, at the junction between its posterior and middle third.
  - Its duct is 2 cm long and opens between hymen and labia minora.
- **Clinically**:
  It appears as cystic swelling of a variable sizes in the posterior part of the labium majus. Secondary infection leads to a painful Bartholin abscess, which is tense, tender, surrounded by vulvar redness, swelling, and oedema.
  [Oral Q: S-Shaped vulva]
- Treatment is by **marsupialization** to create a new opening between the duct wall and the skin.

"This line of treatment is preferable to excision as it is easier, associated with less bleeding and shorter convalescence, together with preservation of the function of the gland. A penrose drain or a small word catheter may be left for a few days to guard against closure of the tract and recurrence of the cyst".

---

3. Bartholin cyst; Diagnosis and treatment (june 2013).

See before.

Vaginal discharge

4. Causes of purulent offensive discharge (june 2009).

(Traumatic-Inflammatory-Neoplastic-Miscellaneous = pregnancy)

- 2 related to pregnancy: septic abortion or puerperal sepsis
- 2 traumatic: foreign body in vagina or neglected pessary
- 2 inflammatory: pyometra or pelvic abscess evacuating vaginally
- Infected neoplasms as submucous fibroid polyp , endometrial , cervical , and vaginal cancers.

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5. DD of vaginal discharge (june 2007)

1. *Normal vaginal discharge*

   White in color, semi fluid in nature, with little or no smell, small in amount < 0.5 ml/day, with a slightly acidic PH (3.8-4.5).
2. *Leucorrhea*
   - **Excessive white non infected vaginal discharge** due to excess of the normal secretion or transudation of the cervix, vagina, and Bartholin's gland) .
   - It may be related to:
     - **Physiologic causes:**
       Mostly related to oestrogen effect with increased vascularity and associated pelvic congestion; as in puberty with onset of menstruation, at the preovulatory and the premenstrual phases of the menstrual cycle, and during pregnancy.
     - **Pathologic causes:**
       Associated with pelvic congestion ; as in pelvic inflammatory disease (PID), fibroids, pelvic and adnexal masses.

3. *Abnormal vaginal discharge:*
    a. Colored offensive discharge: bacterial vaginosis & trichomonas vaginalis.
    b. Muco-purulent discharge: e.g. chronic cervicitis.
    c. Purulent offensive discharge: see before.
    d. Blood stained (sanguineous) discharge: atrophic vaginitis, vaginal and cervical ulcers, cervical erosion, fibroid polyp, and malignancies of the vagina, cervix, and endometrium .
    e. Serous (watery) discharge: intermittent hydrosalpinx , and urinary fistula.
    f. Cottage cheese discharge in candidiasis with severe itching.
    g. Urine: if there is vesico-vaginal fistula.

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6. Give an account on DD of pruritus vulvae.

1. *Pruritus associated with vaginal discharge (80%):*

   Trichomonas vaginalis are the commonest vaginal infestations associated with pruritus. They account for at least 80% of all cases.
2. *Pruritus not associated with vaginal discharge (20%):*
    a. Generalized pruritus: as in cases of jaundice, diabetes mellitus (neuropathy & infection cause pruritus) & uraemia.
    b. Allergy & drug sensitivity: skin sensitivity to various chemical constituents of toilet preparations such as soaps, bath salts and antiseptics may explain some cases. Rarely idiosyncrasy to chemical or rubber contraceptives is present .
    c. Skin diseases not specific to the vulva: as scabies & seborrheic dermatitis.
    d. Chronic epithelial dystrophies: as lichen sclerosus (premalignant).

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7. Clinical approach for a 25 years old patient presenting with vaginal discharge. (June 2017)

History:

1. Recent use of systemic or local antibiotics.
2. Recurrent vulvo-vaginal infections.
3. Vaginal hygienic practices e.g. frequency of douching.
4. Menstrual history: relation of discharge to menses.
5. Obstetric history: relation of the discharge to recent pregnancy, delivery or abortion.
6. Contraceptive history: use of spermicidals, diaphragms and vaginal rings.
7. Sexual history: relation of intercourse to discharge and associated symptoms.
8. History of medical illness as diabetes mellitus & HIV.

Symptoms:

1. Character of the discharge; its amount, color, consistency& odor.
2. Associated burning sensation(the commonest symptom)
3. Presence of itching or pruritus.

Signs on examination:

1. The vulva is inspected for associated vulvitis &/or Bartholinitis.
2. The vagina and cervix are inspected for: white plaques in vaginal candidiasis, Strawberry spots in trichomonas vaginalis vaginitis and grey frothy discharge in bacterial vaginosis.
3. Milking of urethra through the vagina for detection of gonorrhea.
4. Bimanual examination for detection of uterine or adenexal masses.

Lab diagnosis:

1. Wet mount preparations& microscopic examination reveals: clue cells in bacterial vaginosis, hyphae in candidiasis& flagellated trichomonas in trichomonas vaginitis.
2. Addition of 10% KOH may reveal fishy amine odor in bacterial vaginosis.
3. Vaginal swabs for culture will reveal other bacterial causes & Pap smear may reveal trichomonas vaginalis.
4. Biopsy from suspicious vaginal or cervical ulcers or masses.

8. Enumerate benign vulvar neoplasms.

1. Cystic tumors.

2. Solid tumors:

"FPAL and NG (like the sheet obstetric code and nulligravida)"

a. Fibroma
b. Papilloma
c. Adenoma
d. Lipoma
e. Nevus
f. Granulomatous caruncle & caruncle

---

9. Write an account on benign neoplasm of the vulva (June 2017).

1. Solid tumors:

A. Lipoma:

- Arises from the subcutaneous tissue of the vulva.
- Pedunculated & dependant with growth.
- Treatment is by excision.

B. Fibroma:

- Arises from the fibrous tissue of the round ligament & the vulvar connective tissue.
- Pedunculated and firmer than a lipoma.
- On occasions they become sarcomatous.
- Treatment is by excision.

C. Squamous cell papilloma:

- Small single, formed of papillae of stratified squamous epithelium covering a core of vascular connective tissue.
- It should be differentiated from:
  - Condyloma accuminata (caused by HPV) which are usually multiple
  - Fibro-epithelial polyps small, sessile or pedunculated.
  - Bilharzial papillomata.

D. Hydradenoma:

- Arises from the apocrine sweat glands.
- Appears as a small nodule on the labium or interlabial sulcus.
- Diagnosis and treatment is by excisional biopsy.
- Although it is not malignant, the histologic examination of the papillary nature of the tumour **may be mistaken as adenocarcinoma.**

E. *Nevus:*
   - Pigmented nevi of the vulva have functional activity which carries a risk of subsequent malignant transformation.
   - Excisional biopsy should be performed in all pigmented lesions of the vulva, so that the tissues could be sent for histologic examination.
   - Also **they shouldn't be treated with cryo -surgery or LASER therapy so that histologic examination could be performed.**

F. *Caruncle:*
   - Arises from the posterior part of lower end of urethra.
   - Composed of a very vascular stroma **(**that's why they appear red in color) covered with squamous or transitional epithelium & usually infected.
   - The patient is usually an elderly woman complaining of dysuria and bleeding.
   - Treatment is by excision & histologic examination.(malignant change is rare)
   - The base of the tumour on the urethral mucosa should be cauterized.

G. *Granulomatous caruncle:*
   - It's a chronic infection of the periurethral tissue.
   - It's called a caruncle but it is not neoplastic and is often symptomless.
   - **It's even more common than a true caruncle.**
   - Treatment is by cauterization & recurrence is common; as Infection in this area must involve the para-urethral glands& complete cure is difficult.
   - A search for vaginal or bladder source of infection must be made.

H. *Other tumours: hemangioma & neurofibroma.*

2. *Cystic tumours:*
   - Usually benign
   - If large, they are removed by simple surgical excision.

Mnemonic for ttt of vulvar carcinoma (Page 229 Department book):

فيه خطة standard و هنطلع من كل خطوة تعديل عشان نعرف كل الvariations في الplan treatment.

Surgery:

Radical vulvectomy + bilateral groin dissection (LN removal) + Pelvic LN removal.

Instead of a butterfly incision how about **separate incisions (triradiate)?**

Instead of radical vulvectomy, if small and no depth of invasion more than 1 mm how about **local radical excision?**

If it is unilateral how about **unilateral groin dissection**?

Instead of pelvic LN removal how about **Post OP radiotherapy?**

If late stage? PreOP radio and palliative surgery.

7. ONCOLOGY

Premalignant lesions

1. Cervical intraepithelial neoplasia: definition, grades and diagnosis. (june 2010)

Definition:

- Presence of atypical cells inside the cervical epithelium without invasion of the basement membrane.
- The term CIN describes a spectrum of intraepithelial atypical (dysplastic) changes occurring within the squamous epithelium of the ectocervix that carry a premalignant potential.

Grades:

i. *CIN 1:*

Dysplastic cells occupy the **basal one third (1/3**) of the thickness of the squamous epithelium. Cells of the upper 2/3 show normal stratification and maturation.

ii. *CIN II:*

Dysplastic cells occupy **one half (1/2**) of the thickness of squamous epithelium. Cells of the upper 1/2 show normal stratification and maturation.

iii. *CIN III:*

Dysplastic cells occupy **the full thickness** of the squamous epithelium, without invasion of basement members.

Diagnosis:

- CIN usually affects women in younger age groups (25-45 years).
- It is usually asymptomatic.
- Diagnosed through **routine screening** of high risk women by the Pap smear test.
- Suspicious Pap smears are confirmed by colposcopy and biopsies from abnormal areas.

1. *Cervical pap smear:*
   - The pap smear test based on cytological examination of cells shed from both the ectocervix and endocervix.
   - It is performed using cytobrush to wipe cells from endocervical canal and from surface of the TZ of the ectocervix.
   - Cells obtained are spread on a glass slide fixed by ethyl alcohol and stained by **Papanicolaou stain.**
   - The test is done as an office procedure with an **accuracy rate up to 95%**.
   - **Interpretation of pap smear:**
     i. **Negative.**
     ii. **ASCUS** (atypical squamous cells of undetermined significance); this indicates cells that are suggestive but do not fulfil the criteria for squamous intraepithelial lesions **(SIL).** For Its evaluation, testing for **HPV**, **repeat cytology, and colposcopy**, are required.
     iii. **LSILs** (Low grade squamous intraepithelial lesions) concomitant with **CIN I.**
     iv. **HSILs** (High grade squamous intraepithelial lesions) concomitant with **CIN II & CIN III**
     v. **Squamous cell carcinoma**.

vi. **Atypical glandular cells.**

vii. **Endocervical Adenocarcinoma.**

2. *Colposcopy:*
   - Colposcopy allows inspection of the TZ with **a magnification** up to **20 times** after applying **3%-5% acetic acid solution.**
   - Colposcopy directed biopsies are performed from areas of abnormal epithelium (**aceto-white areas, punctuation, mosaicism, leukoplakia**, and **Schiller's iodine negative areas**) with **accuracy of 85%- 95%.**
   - **Endocervical curettage** is performed to rule out dysplasia when the TZ in the cervical canal is not properly visualized in presence of an abnormal pap smear.

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2. Treatment of CIN. (Sep. 2011)

1. *Low grade lesions (LSILs - CIN 1):*
   a. **Conservative** treatment; (treat infection and repeat smear within 12 weeks);
      - 70% will show spontaneous regression.
      - Only 15% may progress to high grade abnormality.
   b. **Destruction** of abnormal cells at TZ if the lesion persists in repeated smears;
      - Ablation using **CO_2 laser.**
      - **Cauterization** by heat or electro-cautery.
2. *High grade lesions (HSILs – CIN II - III):*

   HSILS are managed by **excision** of abnormal cells at the TZ since they are more likely to progress to preinvasive or invasive cancer if neglected or inadequately treated. Excision is performed via:

   a. Cold knife conization:

   The **gold standard** as it yields an adequate clean margin. However postoperative bleeding, infection, cervical stenosis, and cervical incompetence are major complication of this simple procedure.

   b. Loop Electrosurgical Excision Procedure (LEEP):

   A hot metal wire loop is used to excise a wedge of cervical tissue at transformation zone. It has the drawback of yielding a smaller pathologic specimen with charred margins, but is associated with less postoperative complications.

   c. Total abdominal hysterectomy (TAH):

   In older patients with resistant disease and those not desiring fertility.

   Follow up after Treatment: Despite the efficiency of techniques in treating CIN yet recurrence is still common and follow up by **annual Pap smear** is recommended for around **10 years.**

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3. CIN: Grades, diagnosis and treatment (June 2014 / Sep. 2012)

As mentioned above.

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4. CIN: diagnosis and treatment. (June 2015)

As mentioned above.

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5. Premalignant lesions of vulva. (Sep. 2013)

Definition:

- Presence of **atypical (dysplastic cells**) within the vulvar epithelium **without invasion** of the basement membrane.
- VIN seems to have low malignant potential. It commonly occurs in younger age where almost half of the cases are younger than 41 years.

Diagnosis:

Symptoms:

1/3 of cases are asymptomatic, however VIN often presents as **pruritus vulvae.**

Signs:

- Lesions are **often raised** above the surrounding skin **with a rough surface.**
- **Color is variable**: whitish due to hyper Keratinization. 'red due to thinning of epithelium, or dark brown due to melanin deposition in the epithelial cells.
- Lesions are often **multifocal** that is why wide excision is mandatory.
- Painting the vulva with 5% acetic acid will result in VIN areas turning withe and mosaic or punctuations will be visible by the N.E., hand lens or colposcopy. **Biopsies** are taken from acetowhite areas to rule out invasive cancer.

Treatment:

- The youth of many patients, the multifocal nature of the disease, and the discomfort and mutilation of surgical wide excision, makes it necessary to be cautious and **conservative** to avoid making the treatment worse than the disease.
- **Asymptomatic** cases **especially < 50 years** of age are managed **conservatively**, with **repeated biopsies** to exclude progression of the disease.
- **Symptomatic** cases are treated by **topical steroids for 3-6 months** to relieve symptoms.
- If the lesion is **small; excision biopsy** is enough (both diagnostic and therapeutic).
- **Wide** and **multifocal** lesions: **skinning vulvectomy** with or without skin graft is performed.
- **Follow up** and **re-biopsy** are essential to detect invasive disease among those who relapse.

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6. Endometrial hyperplasia: etiology, pathology and diagnosis (june 2012)

Endometrial hyperplasia

Definition:

Thickening of endometrium due to increased size and number of proliferating glands +/- stroma.

Etiology:

Unknown etiology, but multiple risk factors are present:

- Nulliparity (absent periods of amenorrhea during pregnancy and lactation).
- High socioeconomic standard.
- White race.
- Late menopause: age > 55 years (long periods of anovulatory premenopausal cycles).
- PCOS (chronic anovulation results long periods of E2 unopposed by PRG).
- Marked obesity (increased peripheral conversion of androgen to E1 in adipose tissue).
- Unopposed oestrogen therapy (as oestrogen replacement therapy in menopause).
- Long term **Tamoxifen therapy** (in treatment of oestrogen receptor positive breast cancer).

- Oestrogen producing ovarian neoplasm.
- Family history (positive 50%).

Pathology:

Gross picture:

Diffuse endometrial thickening or multiple endometrial polyps.

Microscopic picture (+ malignant potential in %):

- **Simple (cystic) EH (1%):**
  Swiss cheese appearance.
- **Complex (adenomatous) EH (3%):**
  Back-to-back arrangement.
- **Simple EH with atypia (8%).**
- **Complex EH with atypia (29%).**

Diagnosis:

Symptoms:

- Almost is asymptomatic & only diagnosed accidently during routine screening.
- Some cases may present with AUB (postmenopausal).

Signs:

- General: breast examination.
- Abdominal: no signs.
- Local: normal or symmetrically increased uterine size (12 weeks).

Investigations:

1. **Transvaginal sonography (TVS):**
   The most important investigation to detect increased endometrial thickness in postmenopausal women above the cut-off value of **5 mm** is strongly suspicious of EH.
2. **Endometrial biopsy (Curettage):**
   Gold standard to diagnose EH.

Treatment:

1. **Progestin for 3-6 months**
   Action:
   - Anti mitotic
   - Anti estrogen
   - Medical curettage

   Indications:
   - Simple or complex hyperplasia without atypia
   - Simple hyperplasia with atypia
2. **Hysterectomy**
   Indication:
   - Failed progestin
   - Complex with atypia
   - Postmenopausal with any type of atypia

7. Methods of screening of genital cancer in postmenopausal patients. (Sep. 2016)

1. *Screening of cervical cancer: (through cervical pap smear):*
   See before .
2. *Screening of endometrial cancer: (through TVS):*
   Increased endometrial thickness in postmenopausal women above the cut-off value of **5 mm** is strongly suspicious of EH. Thicker endometrium **>10 mm** correlates with EC.
3. *Screening of ovarian cancer:*
   - **Routine yearly pelvic examination** in **premenopausal and postmenopausal** women.
   - **A palpable post-menopausal ovary** must always call for further investigation.
   - **Periodic TVS** coupled with **a serum CA-12S** in those with an **enlarged ovary** have been proposed for screening of ovarian cancer (risk of malignancy scoring system).
   - **Genetic screening**; testing for BRCAl or BRCA2 mutation carriers in women with strong family history of breast and ovarian cancer.
4. *Screening of valval and vaginal cancers:*
   Through Pap smear and colposcopies directed biopsies.

Malignancy of uterine corpus

1. Clinical picture of endometrial carcinoma. (sep 2012)

Clinical picture:

EC presents as AUB in postmenopausal women, with added risk to those who are nulliparous, obese, diabetic and hypertensive

A. Symptoms:

- **Post-menopausal bleeding**: is the commonest presenting symptom.
- **Metrorrhagia**: perimenopausal women with AUB. Bleeding is usually profuse, persistent and recurrent even after medical and hormonal treatment.
- **Other symptoms** include offensive vaginal discharge, menstrual cramps, and deep pelvic pain.

B. Signs:

a. <u>General:</u>

- Cancer corpus triad: PCO
- Cachexia
- Metastasis
- Anemia

b. <u>Abdominal:</u>

Slightly enlarged uterus or mass is noticed if there is fibroid, pyometra

c. <u>Local:</u>

- **<u>PV and speculum:</u>**
  Vaginal and cervical discharge
- **<u>Bimanual:</u>**
  Size, consistency and tenderness on mobility

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2. Treatment of endometrial carcinoma (1997).

Management of EC:

Following surgical staging the treatment will depend upon several factors including: the stage of the disease, which is the most important factor, the histologic type and the grade of the tumour and the patient age and general health.

- *Stage I:*

a. Surgery:

- An exploratory laparotomy with total abdominal hysterectomy and bilateral salpingo-oophorectomy (TAH-BSO) is the **GOLD STANDARD** treatment, performed in all patients, unless there are absolute medical contraindications.
- Peritoneal washings are taken with normal saline for cytological examination.
- Pelvic lymphadenectomy is performed on high risk cases including those with serous, clear all or grade 3 histology tumour and in cases with stage IB (either outer half myometrial involvement or cervical glandular extension).

b. Radiation therapy:

- Cases of adenocarcinoma stage IA or B , grade 1&2 , can be followed without adjuvant radiotherapy.

- High risk cases, with serous or clear cell carcinoma, any grade 3 tumour or with one positive pelvic node, should receive external beam pelvic radiation.
- Cases with multiple pelvic LN of para aortic LN involvement should receive extended field radiation (pelvic and abdominal).
- Patients medically unfit for surgery may receive radiation therapy alone. A combination of intracavitary and external beam radiation, with an overall 5-year survival rates 20% lower than treated by TAH-BSO.

c. *Hormone therapy:*

- EC rarely occurs in women < 40 years of age. These tumours are usually early stage, and low-grade, and there is frequently a desire to preserve fertility and to avoid hysterectomy.
- High dose **medroxyprogesterone acetate** (200 mg twice daily) for 3-6months will reverse the changes in about 2/3 of cases. Recurrences are common.

- *Stage II:*

  When the cervix involved, a radical hysterectomy with pelvic lymphadenectomy will be the procedure of choice in surgically fit patients.
- *Management of advanced cases (stage III & IV)*

  It may include combination of:
  - TAH-BSO: if possible as palliative therapy for bleeding, pain & other pelvic symptoms.
  - Radiation therapy: extended external beam radiation.
  - Chemotherapy: combination of **cisplatin** and **Doxorubicin** either alone or with radiotherapy.
- *Recurrent cases:*
  - More than 80% of recurrences occur within the first 2 years of treatment.
  - The vaginal vault is the primary site for recurrent disease.
- *Other sites include: the pelvis, upper abdomen or distant metastases.*
  - The treatment of recurrent cases depend on the site and extent of recurrence.
  - **Progesterone** may help only in well differentiated tumours containing oestrogen receptors.
  - **Radiotherapy and/or chemotherapy** may be tried in less differentiated tumours.
  - **Limited surgery** is rarely indicated.

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3. Surgical staging and treatment of endometrial carcinoma (june 2013, sep 2014).

Staging of EC:

Stage	Characteristics
Stage I	**Tumour confirmed to the corpus uteri**
IA	No or < ½ myometrial invasion
IB	½ or > ½ myometrial invasion
Stage II	**Tumour invades cervical stroma, but does not extend beyond the uterus.**
Stage III	**Local and/or regional spared of the tumour**
IIIA	Tumour invades serosa of the corpus uteri and/or adnexa
IIIB	Vaginal and/or parametrial involvement
IIIC	Metastases to pelvic (IIIC1) and/or para aortic LN (IIIC2)

Stage IV	**Tumour invades bladder or bowl mucosa and/or distant metastases**
IVA	Tumour invades bladder or bowl mucosa.
IVB	Distant metastases include intra-abdominal metastases and/or inguinal LN.

Treatment:

See before.

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4. Diagnosis & management of endometrial carcinoma (sep 2007)

Diagnosis:

i. Clinical picture:

See before.

ii. Investigations:

1. Special investigations:

a. Transvaginal sonography (TVS):

- Increased endometrial thickness in postmenopausal women above the cut-off value of 5mm is strongly suspicious pf EH. Thicker endometrium>10mm correlates with EC.
- Associated uterine lesion as myomas, polyps, hematometra, or pyometra.
- Associated adnexal masses: as ovarian neoplasm.

b. Fractional curettage (FC):

- FC the GOLD STANDARD method for diagnosis of EC.
- An endocervical biopsy is first obtained before cervical dilatation.
- Full curettage of endometrial cavity (isthmus, anterior and posterior walls, fundus and uterine cornu). This is important to confirm the diagnosis of EC, its histologic type and grade and for detection of spread to the cervix.

c. Pap smear test:

A pap smear has an accuracy of <50% in diagnosis of EC, hence considered unreliable when compared with endometrial biopsy.

2. Other investigations:

Preoperative work up including:

- Chest x-ray to exclude lung metastasis.
- Abdominal ultrasound to exclude liver metastases, abdominal masses and ascites.
- CT scan for pelvis and abdomen, may point to LN involvement.
- MRI is sensitive in assessment of depth of myometrial invasion if present.

iii. Staging:

See before.

Management:

See before.

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5. Risk factors, pathology & treatment of endometrial carcinoma. (jun. 2014)

Risk factors:

- Nulliparity (absent periods of amenorrhea during pregnancy and lactation).
- Late menopause: age > 55 years (long periods of anovulatory premenopausal cycles)
- PCOS (chronic anovulation results long periods of E2 unopposed by PRG)
- Marked obesity (increased peripheral conversion of androgen to E1 in adipose tissue)

- Unopposed oestrogen therapy (as oestrogen replacement therapy in menopause)
- Long term **Tamoxifen therapy** (in treatment of oestrogen receptor positive breast cancer)
- Atypical endometrial hyperplasia
- Oestrogen producing ovarian neoplasm.

Pathology:

1. *Gross pathology:*
   a. Localized type: localized area of endometrial affection in the form of nodule or polyp.
   b. Diffuse type: diffuse endometrial thickening or multiple endometrial polyps.
2. *Microscopic histopathology:*
   a. Adenocarcinoma: is the commonest pathological type of EC and carries best prognosis.
   b. Adenoacanthomas: is adenocarcinoma with areas of benign squamous metaplasia. It carries some prognosis as adenocarcinoma.
   c. Adenosquamous: is adenocarcinoma with areas of malignant squamous carcinoma. It carries poor prognosis than adenocarcinoma and adenoacanthoma.
   d. Primary squamous cell carcinoma, papillary serous carcinoma, and clear cell carcinoma: are particularity aggressive but very rare.
3. *Grading of the tumour:*

   Determined by degree of abnormality of glandular architecture and degree of nuclear hyperplasia:
   a. Well differentiated tumour (grade I): normally looking glandular epithelium <5% solid structure. They are slowly invasive and carry the best prognosis.
   b. Moderately differentiated (grade II): glandular structure mixed with papillary and occasional solid areas<50%. It is more aggressive and carries intermediate prognosis.
   c. Poorly differentiated (grade III): the structure becomes predominantly solid, with minimally identified glandular epithelium. The tumour is highly aggressive with early deep myometrial invasion and poor prognosis.
4. *Spread of EC:*

   i. Direct spread:
   a. Endometrium
   b. Myometrium
   c. Downwards to cervix
   d. Ovary and peritoneum
   e. Upwards : peritonitis

   ii. Lymphatic:
   1. **Fundus:**
      Through ovarian artery to paraaortic lymph nodes
   2. **Body:**
      Uterine then internal iliac artery to internal, external and common ilac lymph nodes
   3. **Isthmus and lower body:**
      Paracervical nodes
   4. **Cornu:**
      Through round ligament to inguinal L.N

5. **<u>Ovary and vaginal vault involved in lymphatic spread</u>**

iii. *Vascular spread: occurs late with higher grade tumour.*

- Metastases to nearby intra-pelvic organs.
- Distant extra-pelvic metastases rarely occur (liver, lung, brain & bones)

Treatment of EC:

See before.

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6. Choriocarcinoma: incidence, pathology & spread.

Incidence:

More common to affect women of low socioeconomic standards.

- **50%** of cases will develop following evacuation of a molar pregnancy .
- **25%** following abortion.
- **25%** following term pregnancy .
- **5%** following an ectopic gestation .

Pathology:

A. *Gross:*
   - Tumor appears as a friable hemorrhagic nodular mass arising in the body of the uterus, 1st invading the endometrium, projecting into the cavity, then extending to invade the myometrium.
   - The ovaries may be enlarged showing multiple theca lutein cysts.

B. *Microscopic:*
   - The tumor is comprised of sheets of sheets of anaplastic cytotrophoblasts & syncytiotrophoblast cells with prominent haemorrhage, necrosis & vascular invasion.
   - There are no chorionic villi.

C. *Spread:*
   - <u>Hematogenous spread :</u>

     Early & widespread metastases to various sites , namely to the lungs (80%) , the vagina (30%) , liver (30%) , CNS & brain (10%) .
   - <u>Direct spread :</u>

     Malignant cells may directly invade the myometrium .

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7. Diagnosis, special investigations & treatment of choriocarcinoma (jun.2011)

Diagnosis:

i. *Symptoms:*

   Persistent vaginal bleeding that continues for > 6weeks following molar evacuation, abortion or term pregnancy.

ii. *Signs:*
   - Bimanual vaginal examination may reveal a slightly symmetrically enlarged uterus that may be soft in consistency.
   - In cases with vaginal metastases, a soft haemorrhagic nodule may be seen within the vagina.
   - Cysts can be felt (theca lutein).

iii. Special investigations:

A. Elevated serum B-HCG levels:

- Persistently high B-HCG levels after evacuation of a molar pregnancy in absence of new pregnancy is the GOLD STANDERD in diagnosis of gestational choriocarcinoma.
- Biopsy is not needed to establish diagnosis and start treatment.
- Cases are classified as high risk or low risk cases according to serum B-HCG levels so treatment is planned based on such levels.

B. TVS may show:

- Slightly symmetrically enlarged uterus with absence of intrauterine gestational sac.
- A small mass within endometrial cavity that may extend to myometrium.
- Colour doppler indices to the mass may be suggestive of malignancy (low RI and high diastolic flow due to associated neo-vascularization).
- Bilateral theca lutein cysts of the ovaries.

C. MRI:

Confirms the lesion and help in evaluation of intra-myometrial invasion if present.

D. CT:

May be required to exclude metastases in liver, lung and brain.

E. D&C biopsy:

- Biopsy will reveal anaplastic trophoblastic tissue with absence of villous pattern.
- Histological diagnosis is not essential to confirm diagnosis or to start treatment in presence of specific tumour marker B-HCG.

Treatment of choriocarcinoma:

i. Chemotherapy:

Chemotherapy is indicated in all cases of gestational choriocarcinoma. It is considered the main treatment even in cases may require surgical treatment.

A. Single agent:

- **Methotrexate:**
  - MTX is single agent drug of choice for non-metastatic low risk cases, with complete remission rates 0f 60-80%
  - MTX is given as 50 mg dose IM every 48 hours / 4doses.
  - MTX/Folinic acid regimen: where folinic acid is taken orally 30 hours after MTX injection to minimize side effects of MTX.
  - Cycle regimen is repeated after 14 days till negative B-HCG IS obtained.
  - Side effect of MTX include: nausea, vomiting, stomatitis, GIT ulcers, myelosuppression, hepatotoxicity, nephrotoxicity and alopecia.
- **Actinomycin:**

  Is also an effective single agent chemotherapy.

B. Combined agents:

MTX, Estoposide and Actinomycin: is the combination of choice for metastatic high-risk cases and for resistant to MTX single agent chemotherapy.

Follow up after chemotherapy:

Follow up by repeated serum B-HCG is recommended after chemotherapy is completed until negative B-HCG is achieved for 3-6 months.

ii. Surgical treatment:

- Surgery via **TAH** in choriocarcinoma is reserved only for selected cases include:
  - Elderly, multiparous, high-risk cases (>40 years)
  - Cases resistant to combination chemotherapy.
  - Cases complicated by severe haemorrhage, perforation or infection.
- TAH is usually preceded and followed by chemotherapy: MTX 10 mg given at day of operation and continued postoperative for 4 to 5 days to prevent the risk of development of distant metastases.
- There is no need to remove metastases as ovarian metastases is rare is rare can effectively treated by MTX.

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8. Leiomyosarcoma: pathology, clinical picture & treatment.

Pathology :

- Macroscopically: cut surface shows areas of haemorrhage & necrosis.
- Microscopically: spindle shaped or rounded cells, many of them being pleomorphic, with little stroma & primitive blood vessels.
- Histological diagnosis of malignancy: depends on the number of mitoses per high power field (HPF). Patients with > 10 % mitosis per HPF are regarded as having malignant disease.

Clinical picture:

- Symptoms :

  AUB especially in perimenopausal or menopausal women.
- Signs :

  No specific signs , where most cases are diagnosed accidently only after surgical removal at hysterectomy . leiomyosarcoma **is highly suspected in** :
  a. Rapid growth of myomas at any age.
  b. Any growth of myomas after menopause.
  c. Rapid recurrence after myomectomy.
  d. Absence of plane of cleavage during myomectomy.

Treatment:

- TAH BSO is the treatment of choice, followed in many cases by external radiotherapy.
- In general, prognosis is poor , except for early cases of leiomyosarcoma arising in a myoma without associated blood stream spread.

Malignancies of uterine cervix

1. Diagnosis & treatment of cervical carcinoma.

Clinical picture :

Symptoms:

1. *It may be asymptomatic in early stages.*
2. *Contact bleeding:*
   The commonest presentation (postcoital bleeding or bleeding on touch).
3. *Metrorrhagia, or postmenopausal bleeding:*
   The second most common presentation.
4. *Vaginal discharge:*
   Excessive bloody or malodorous.
5. *Pain:*
   Deep pelvic pain and loin pain may be associated with advanced disease.

Signs:

1. *General examination:*
   - Early stages do not affect the patient's general condition.
   - Advanced stages are associated with; chronic blood loss, urinary manifestations and ureteric obstruction which may lead to severe anaemia, uraemia and cachexia.
2. *Inspection via a speculum vaginal examination:*
   - Early stages may show a small friable warty mass, nodule or an ulcer that bleeds on touch.
   - Later on, the mass or ulcer will extend to the vaginal walls obliterating the vaginal fornices.
3. *Palpation by digital vaginal examination (PV):*
   - Early stages show a mass or ulcer that bleeds profusely when touched by the finger.
   - As the disease progresses, the cervix loses its mobility, becomes fixed, and the surrounding parametrium becomes tender and indurated.
4. *Bimanual pelvic examination:*
   The uterus is usually normal in size, except if pyometra develops causing symmetrical uterine enlargement.
5. *Per rectum examination (PR):*
   To evaluate possible parametrial extension and uterosacral involvement.

Complications (spread):

1. *Direct spread:*
   To adjacent tissues including the body of the uterus, the parametrium, upper and lower vagina, and uterosacral ligaments. Later spread may involve the bladder and rectum.
2. *Lymphatic spread:*
   Usually follows direct spread, but may coincide with it. It involves;
   a. <u>Primary groups:</u>
      Include spread from the cervical lymphatics along the paracervical lymph tract,to the obturator (interiliac) , to the external iliac nodes, and occasionally backwards to the internal iliac group (hypogastric).

b. Secondary groups:

Involve drainage into the common iliac and lateral sacral groups. Ultimately the common iliac group drains into the para-aortic lymph nodes.

3. *Blood stream spread:*

Least common, usually in late or advanced cases. Metastases may occur to distant organs as to the liver, lungs, brain and bone.

Differential diagnosis: (of contact bleeding)

See before (bleeding)

Investigations:

Histopathologic examination of ***cervical tissue biopsies*** containing the abnormal epithelium is the gold standard for diagnosis of cervical cancer. Biopsies are obtained as follows:

1. Knife biopsies.
2. Colposcopic Directed Biopsies.
3. Cone Biopsy.
4. Fractional Curettage (FC).

Treatment:

- **Choice of treatment**, whether surgery, radiotherapy or both, and the extent of each of them is based namely on the stage of the disease, grade of the tumour, age and general condition of the patient.
- **Early invasive cancer cervix;** Either **surgery** or **radiotherapy** can be used with comparable results, however surgery is more preferred as it allows for complete resection of the tumour and provides a specimen available for ensuring the extent of the disease. **Chemotherapy** may be also used as a radiation sensitizer.
- **In more advanced stages;** the high morbidity of extensive surgery and possible distant spread of the disease are in favour of choosing the option of **radiotherapy.**

1. *Surgery:*

Surgery is usually the first line of treatment whenever the disease is confined to the cervix (stage IA-IB). If the disease has reached the upper vagina without parametrial involvement (stage IIA) surgery will be as effective as radiotherapy but with less side effects and morbidity. Surgery is superior to radiotherapy in providing a specimen for histopathologic examination that allows evaluation of the safety margin and detects the extent of nodal affection if present.

- **Wertheim's radical hysterectomy** is the standard surgical procedure for invasive carcinoma of the cervix. It involves a ***TAH-BSO + pelvic lymphadenectomy*** *+ removal of* ***paracervical tissue*** *and 2-3 cm from the* ***upper vaginal cuff****.*
- Pelvic lymphadenectomy include removal of; the external, internal, and common iliac nodes, together with obturator and presacral nodes.
- If surgery results in removal of the whole tumour, with a *free safety margin* of healthy tissue and *negative lymph nodes*, there will be usually no further need for postoperative radiotherapy.
- Patients with positive nodal affection or non free safety margin are best offered **adjuvant radiotherapy treatment.**
- The ovaries may be preserved in younger women to benefit fro their hormonal function.
- Schauta's vaginal hysterectomy and lymphadenectomy is rarely resorted to owing to the limited access to pelvic nodes via such procedure ending in incomplete lymphadenectomy.
- The principal complication seen with surgery is related to some degree of bladder

dysfunction due to division of the parasympathetic nerve supply to the bladder that runs within the uterosacral ligaments.

- **Five years survival rates after surgery alone** ranges 75%-100% in stages from IA to IIA. Postoperative radiotherapy with or without chemotherapy may improve survival in some cases.

2. *Radiotherapy:*

Radiotherapy can be used in all stages of cervical cancer either for curative or palliative intent.

- In early stages (I-IIA) cure rates are comparable to those for surgery.
- In more advanced stages (IIB-III) cancer will not be curable by surgery alone, and radiotherapy becomes the preferred first line of treatment. It may be given alone, or in combination with surgery or chemotherapy.
  - **Primary therapy;** usually involves external beam radiotherapy (EBRT) to the pelvis followed by intracavitary treatment or brachytherapy.
  - **Adjuvant therapy;** administered after radical hysterectomy to high risk cases (positive safety margins, and or positive nodal involvement)
- **The five years survival rates for radiotherapy alone** are comparable for survival with surgery alone for stages IA-IIA disease.
- For advanced stages localized within the pelvis, 5- years survival ranges from 50%-80%. For metastatic disease outside the pelvis, survival is less than 15%.
- **Complications of radiotherapy** include, diarrhoea, radiation induced menopause, and variable degrees of vaginal narrowing and fibrosis, few weeks after treatment is completed. Rarely radiation vesico-vaginal fistulas may occur and are difficult to deal with.

3. *Chemotherapy:*

Chemotherapy has proved to play a role as a radiation sensitizer in cases of cancer cervix (e.g.; weekly I.V. Cisplatin), with a nearly 10% increase in cure rates.

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2. Screening & diagnosis of cervical carcinoma.

Screening:

CIN usually affects women in younger age groups (25-45 years). It is usually asymptomatic, accidentally diagnosed through ***screening*** of high risk women by the ***Pap smear*** test, and confirmed by performing ***colposcopy and biopsies*** in women with abnormal smears.

1. *Cervical smears (Pap smear test):*

- The Pap smear test is based on ***cytologic examination*** of cells shed from the ectocervix. It is performed using a **cytobrush** to wipe cells from the endocervical canal, and an **Ayre wooden spatula** to wipe cells from the surface of the ectocervix. Cells obtained are spread on a glass slide fixed by ethyl alcohol and stained by ***Papanicolaou stain***. The test is done as an office procedure with an accuracy rate > 80%, however it carries a small percentage of both false positive and false negative results (15-25%).
- Interpretation of a Pap smear will include :
  - **a. <u>Benign changes</u>**
  - **b. <u>Reactive changes:</u>**
    - Low grade intraepithelial changes LISLs (CIN I).
    - High grade intraepithelial lesions HISLs (CIN II-III).
  - **c. <u>Malignant cells present</u>**

2. *Colposcopy:*
    - Colposcopy allows inspection of the TZ with a magnification up to 20 times after applying 3%-5% acetic acid solution.
    - **Colposcopic directed biopsies** are performed from areas of abnormal epithelium (aceto-white areas, punctuation, mosaicism, leukoplakia, and Schiller's iodine negative areas) with accuracy of 85%- 95%.
    - **Endocervical curettage** is performed to rule out dysplasia when the TZ in the cervical canal is not properly visualized in presence of an abnormal pap smear.

Frequency of cervical cytology screening:

- High risk population should be screened annually within 3 years from onset of sexual activity.
- Women above the age of 30, with low risk factors and 3 consecutive annual negative pap smears should be screened every 3 years .
- Women over the age of 70 with 3 negative Pap smears in the last decade, can consider discontinuation of Pap testing at the physician's advice.

Diagnosis:

See before.

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3. Risk factors of cervical cancer.

1. Early exposure to 1st intercourse , especially with Multiple Sexual Partners.
2. Viral agents as HPV & HSV II.
3. Untreated HSILs which may gradually progress to invasive cancer.
4. Cigarette Smoking due to hypoxia of endocervical cells.

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4. Cervical biopsies: types & values.

See Q1.

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5. Lymphatic spread of cervical cancer.

Usually follows direct spread, but may coincide with it. It involves;

1. *Primary groups:*
   Include spread from the cervical lymphatics along the paracervical lymph tract, to the external iliac nodes, and occasionally backwards to the internal iliac group.
2. *Secondary groups:*
   Involve drainage into the common iliac and lateral sacral groups. Ultimately the common iliac group drains into the para-aortic lymph nodes.

Non-neoplastic ovarian swellings

1. Enumerate non neoplastic cysts of ovary.

1. Follicular cysts
2. Corpus luteum cysts
3. Theca lutein cysts
4. Endometriotic cysts
5. Inflammatory cysts
6. Germinal inclusion cysts

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2. Mention 3 symptoms of benign ovarian cysts and 3 measures for treatment.

Benign Ovarian cysts may include: non neoplastic cysts or benign ovarian neoplasms.

i. Non neoplastic ovarian cysts:

Symptoms:

1. **Asymptomatic:** Follicular cysts are usually asymptomatic.
2. **Menstrual disturbance:** Delayed menstruation or irregular bleeding due to persistent oestrogen production leading to endometrial hyperplasia
3. **Pain (rarely acute abdomen):** If the cyst is large, rapidly growing, or ruptures. It may cause pain in one of the iliac fossae that may simulate that of appendicitis

Treatment:

1. **Conservative treatment:**
   - Small non complicated functional cysts of the ovary are managed conservatively since spontaneous regression is the rule in most of these cysts
   - During the few weeks of follow up US is used to monitor complete cyst resolution
2. **Medical treatment:**
   - Gestagens and OCP may accelerate regression of follicular, CL, and theca lutein cysts
   - GnRH agonists, Gestagens, and OCPs may help minimizing rate of growth of endometriotic cysts thus prolonging the period of conservative management and avoiding or delaying the need for surgical intervention
   - Antibiotics with specific regimens according to CDC recommendation will allow shrinkage in size of inflammatory cysts to control the symptoms and size, and avoid the need for surgical intervention
3. **Surgical treatment:**
   - Large cysts > 7.0 cm, complicated cysts (by haemorrhage or torsion), and persistently growing ones (like endometriomas) are managed surgically from the start or after an adequate interval or trying conservative approach and/or medical treatment
   - Laparoscopy is the preferable approach suitable for most cases.
     Operations include:
     - Undoing torsion of cyst before it becomes ischemic or gangrenous
     - Ovarian cystectomy for cysts that reaches large size or starting complication
     - Ovariotomy (removal of the ovary) is rarely an option, only in cases with severe haemorrhage or gangrene in which ovarian preservation becomes impossible

ii. Benign ovarian neoplasms:

Symptoms:

1. **Abdominal swelling:** If large
2. **Lower abdominal pain & heaviness:**
   - Acute pain if complicated
   - Chronic dull aching if large
3. **Pressure symptoms:**
   - Epigastric pain and dyspnea
   - Frequency of micturition, retention of urine

Treatment:

Surgical removal of the tumour either via:

1. **Ovarian cystectomy:**
   - Consists of shelling out or enucleation of the cyst with preservation of the ovary.
   - Indicated in young patients particularly with bilateral cysts with small size.
2. **Oophorectomy:**
   - Removal of the whole tumour together with the ovary.
   - Indicated when cysts are large can't be removed via cystectomy because of:
     - Difficulty in enucleation of the cyst without rupture and dissemination of its contents together with absence of adequate healthy ovarian tissue to be left.
     - Fear of malignancy especially in larger, bilateral and solid tumours.

"Both can be done either via laparoscopy or laparotomy however cystectomy is better done via laparoscopy except in cases of dermoid cyst carries a high risk for dissemination due to rupture And oophorectomy is better done via laparotomy".

3. **Pan hysterectomy:**
   - Consists of total abdominal hysterectomy with bilateral salpingo-oophorectomy.
   - Indicated if the patient is pre-menopausal and has completed her family.

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3. Corpus luteal Cyst; definition, pathology, diagnosis, and treatment.

Definition:

They are less common than follicular cysts, arising from excessive haemorrhage inside the corpus luteum during the stage of vascularisation. Spontaneous regression and *complete resolution* is almost the rule. *Rarely rupture* with lower abdominal pain and intra-peritoneal haemorrhage may occur.

Pathology:

Cysts are usually unilateral, single, unilocular, small sized (3-7 cm), containing either bloody fluid or clear content. The cyst wall is lined by ***leutinized granulosa cells*** that may continue to secrete *progesterone* causing menstrual disturbance.

Diagnosis:

i. Symptoms:

1. **Menstrual disturbance:** may be present in the form of a short delay in the menstrual cycle, due to persistent progesterone production, commonly followed by irregular vaginal bleeding.
2. **Acute lower abdominal pain:** may be present in one of the iliac fossae if the cyst is complicated with haemorrhage or rupture. (DD; from ectopic pregnancy, where a B-hCG will be positive, and acute appendicitis if on the right side, where a CBC will be

suggestive)

ii. Signs on bimanual examination:

1. Tenderness at mid-ovarian point in the iliac fossa.
2. Tenderness and fullness at one vaginal fornix.
3. A tender cystic mass may be felt in thin patients.

iii. Investigations:

1. Pelvic TAS or TVS. (Gold standard)
2. Laparoscopy is conclusive whenever diagnosis is in doubt or cyst complicated

DD:

1. Follicular cyst and simple serous cyst.
2. Causes of pain in right iliac fossa (appendicitis, ectopic pregnancy, ..).

Treatment:

i. Conservative treatment:

- Small non complicated functional cysts of the ovary are managed conservatively since spontaneous regression is the rule in most of these cysts
- During the few weeks of follow up US is used to monitor complete cyst resolution.

ii. Medical treatment:

- Gestagens and OCP may accelerate regression of the cysts.

iii. Surgical treatment:

- Large cysts > 7.0 cm, complicated cysts (by haemorrhage or torsion), and persistently growing ones are managed surgically from the start or after an adequate interval or trying conservative approach and/or medical treatment
- Laparoscopy is the preferable approach suitable for most cases.
  Operations include:
  - Undoing torsion of cyst before it becomes ischemic or gangrenous
  - Ovarian cystectomy for cysts that reaches large size or starting complication
  - Ovariotomy (removal of the ovary) is rarely an option, only in cases with severe haemorrhage or gangrene in which ovarian preservation becomes impossible

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Benign ovarian neoplasms

1. Clinical picture, complications & treatment of benign ovarian tumour.

Clinical Picture: (symptoms, signs, investigations):

A. Symptoms:

AA PBP

1. ***A**symptomatic:*
   Discovered incidentally during routine examination
2. ***A**bdominal swelling:*
   If large
3. *Lower abdominal **P**ain & heaviness:*
   - Acute pain if complicated
   - Chronic dull aching if large
4. ***P**ressure symptoms:*
   - Epigastric pain and dyspnoea (لفوق)
   - Frequency of micturition, retention of urine (لتحت)
5. ***B**leeding and menstrual disorders:*
   If functional tumor

B. Signs:

Depend on size

Small *(bimanual examination)*	*Large* *(abdominal examination)*
Felt on one side of the uterus as round, smooth, mobile and usually cystic movement of mass not transmitted to the cervix	- **Inspection:** Symmetrical abdominal enlargement - **Palpation:** Abdominal mass with well-defined upper and lateral borders, smooth or lobulated surface - **Percussion:** Central dullness with resonant flanks

In excessively large ovarian cysts, cachexia may occur due to rapidly growing tumor.

Investigations:

تشوف بأشعة/ تشوف بعنيك/ تشوف تحاليل

1. *US:*
   - Gold Standard
   - Differentiates between benign and malignant
2. *Tumour markers:*
   - CA125 elevation → epithelial ovarian cancer but can be elevated with endomeriomas
   - CA19-19 elevation → mucinous carcinoma
3. *Laparoscopy:*
   - To see any adnexal mass
   - To differentiate between → ovarian cyst and tubo-ovarian cystic masses
     → solid ovarian Fibroma & pedunculated sub-serous myoma

4. *If there is pressure symptoms do I.V.P.*

Complications:

(H/R/T/in/in/malignant transformation)

Hemorrhage/**R**upture/**T**orsion/**In**fection/**In**carceration/**Malignant transformation**

1. *Hemorrhage:*

Aetiology:

- **T**orsion or **T**rauma
- Spontaneous during pregnancy

Clinical picture:

Acute abdomen; **T**ense and **T**ender

Treatment:

Ovariotomy by laparotomy

2. *Rupture:*

Aetiology:

- **T**rauma, **T**orsion
- Haemorrhage
- During labor or rough vaginal examination

Clinical picture:

- Picture of acute abdomen:
- Severe pain, abdominal rigidity, tenderness and rebound tenderness.
- If it was a dermoid → severe chemical peritonitis
- If it was mucinous cystadenoma → pseudo-myxoma peritonii
- If internal haemorrhage occurred → hypovolemic shock (tachycardia, hypotension, oliguria, ….)

Treatment:

- Sedatives to alleviate pain.
- Resuscitation of the patient to restore BP
- Ovariotomy: as an emergency procedure usually via laparotomy and peritoneal lavage to remove contents of the cyst.

3. *Torsion:*

Factors predisposing to torsion:

- Moderate sized tumours with long pedicle (BCT is the commonest tumour to undergo torsion)
- Free mobility with absence of surrounding adhesions.
- Pregnancy and puerperium: due to displacement of the tumour during pregnancy and laxity of abdominal wall after delivery.

Clinical picture:

Acute abdomen

Treatment:

- Ovariotomy → if gangrenous and no viable tissue
- Ovarian cystectomy → if adequate viable healthy tissue is still there.

4. *Infection:*

Aetiology:

- In the puerperium (following abortion or labour)
- From a nearby infected organ
- Or following torsion

Clinical Picture:

- Fever, tachycardia, malaise
- By bimanual examination: rapid increase in size, fixed tender tumour

Treatment:

- Antibiotics (broad spectrum/ anaerobic)
- Ovariotomy via laparotomy

5. *Incarceration:*

Definition:

Impaction in Douglas' pouch

Clinical Picture:

Pressure symptoms on bladder and rectum

Treatment:

Surgical removal of the tumour

6. *Malignant Transformation:*
    - More in solid tumours than cystic
    - Papillary serous cystadenoma (50%)
    - Mucinous cystadenoma (5%)
    - Benign cystic teratoma (1%)

Treatment:

Surgical removal of the tumour either via:

1. *Ovarian cystectomy:*
    - Consists of shelling out or enucleation of the cyst with preservation of the ovary.
    - Indicated in young patients particularly with bilateral cysts with small size.
2. *Oophorectomy:*
    - Removal of the whole tumour together with the ovary.
    - Indicated when cysts are large can't be removed via cystectomy because of:
        - Difficulty in enucleation of the cyst without rupture and dissemination of its contents together with absence of adequate healthy ovarian tissue to be left.
        - Fear of malignancy especially in larger, bilateral and solid tumours.

    "Both can be done either via laparoscopy or laparotomy however **cystectomy is better done via laparoscopy** except in cases of dermoid cyst carries a high risk for dissemination due to rupture and **oophorectomy is better done via laparotomy**".
3. *Pan hysterectomy:*
    - Consists of total abdominal hysterectomy with bilateral salpingo-oophorectomy.
    - Indicated if the patient is pre-menopausal and has completed her family.

2. Benign cystic teratoma of ovary; mention incidence, complication and treatment

Incidence:

- 50% of ovarian neoplasms below age of 20
- Bilateral in 12% of cases
- Usually of moderate size

Complications:

See before (but write **TORSION** at first as it's the most common one)

Treatment:

Surgical Removal:

A. Of cyst (cystectomy):
   - Enucleation with preservation of ovary
   - Indicated in young patient, with bilateral small sized tumours

B. Of ovary (oophorectomy):
   Removal of the whole tumour together with the ovary

C. TAH BSO:
   If premenopausal and completed her family

N.B.

Laparoscopy is less good than laparotomy as laparoscopy carries a high risk for dissemination due to rupture

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3. Complications and treatment of benign ovarian tumours.

See Q1

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4. Rupture of ovarian cyst as a complication of benign ovarian tumours.

See Q1 (point 2)

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5. Complications of benign ovarian tumours.

See Q1

Malignant ovarian neoplasms

1. **Clinical features suggestive of malignancy of ovarian tumors.**

A. *History and symptoms:*

- Tumours discovered at **extremes of age** (prepubertal period, young girls, and postmenopausal) are highly suspicious for malignancy.
- Tumours discovered in women with **family history** of breast, colonic or ovarian cancer.
- Tumours associated with rapid **weight loss**, rapid progressive abdominal enlargement and persistent GIT symptoms.
- Functioning tumours with **feminizing** or **virilising** effects.

B. *General examination:*

- Malignant cachexia (with marked and rapid weight loss and dehydration).
- Palpable supraclavicular lymph nodes especially on the left side (Virchow`s glands).
- Pleural effusion, however it may be present in Meig`s syndrome.
- Presence of an associated breast mass on breast examination.
- Unilateral lower limb oedema (unilateral pressure with venous and lymphatic obstruction).

C. *Abdominal examination:*

- Inspection: Abdominal enlargement with overlying skin showing Peau d`orange.
- Palpation: Tumour which is Solid (or partially solid), fixed especially bilateral.
- Percussion: Ascites except ovarian fibroma in Meig`s syndrome.

D. *Pelvic examination:*

- Nodules in Douglas pouch in the presence of non-tender adnexal mass.
- Bilateral, especially if solid adnexal masses are very presumptive.
- Fixed pelvic masses especially if amalgamated with pelvic organs (Frozen pelvis).

E. *US criteria suggesting malignancy:*

- Heterogenous echopattern
- Intracystic and extracystic papillae
- Bilaterality
- Presence of ascites
- Low resistance Doppler flow of the tumor vessels

F. *At laparotomy:*

- Ascites, especially if altered blood stained ascites.
- Bilaterally, fixation and invasion of the capsule.
- Extra-cystic papillae and adhesions to surrounding structure.
- Peritoneal nodules or secondary deposits in omentum, intestine, liver or lymph nodes.
- Variable consistency with a cut section of the tumour shows haemorrhage and necrosis.

2. Primary epithelial ovarian cancer; operative staging and surgical treatment.

Operative staging:

Performed through an exploratory laparotomy; via midline sub-umbilical suprapubic abdominal wall incision in which the following is performed:

1. Exploration of the pelvic and peritoneal cavity to assess whether the tumour is confined to one or both ovaries, or extending to other pelvic or abdominal organs.
2. Aspiration of any ascetic fluid present (or perform saline peritoneal washing, if no ascites present) for cytological examination to detect malignant cells in the peritoneal cavity.
3. Performing a TAH-BSO, together with infra-colic omentectomy, and pelvic and para-aortic lymph node sampling, whenever possible.
4. Resection of any visible enlarged nodules or masses in pelvic or abdominal cavities or any pelvic or extra-pelvic tumour masses > 2cm (debulking or cytoreductive surgery).

Staging:

I: Ovary

IA: Limited to one ovary (capsule intact).

IB: Both ovaries (capsule intact).

IC: One or both ovaries with the following:

IC1: Surgical spill intra operatively.

IC2: Capsule ruptured.

IC3: Malignant cells in ascites or peritoneal washing.

II: Pelvic extension

IIA: Uterus- fallopian tube- ovaries.

IIB: Other pelvic intra peritoneal tissues (Serosa).

III: L.N.s + abdomen

IIIA: Retroperitoneal L.N.s +/- microscopic peritoneal involvement.

IIIA1: +ve retroperitoneal L.N.s only

IIIA1 (i): Metastasis < or = 10 mm.

IIIA1 (ii): Metastasis > 10 mm.

IIIA2: Microscopic extra pelvic involvement +/- +ve retroperitoneal L.N.s.

IIIB: Macroscopic peritoneal metastases < or = 2cm +/- L.N.s.

IIIC: Macroscopic peritoneal metastases > 2cm +/- L.N.s.

IV: Distant metastasis

IVA: Pleural effusion with +ve cytology.

IVB: Extra abdominal organs.

Surgical treatment:

1. *Early stage ovarian cancer:*
   - **A. <u>TAH-BSO and infra-colic omentectomy:</u>**
     - Is the standard treatment for patients with stages (I-IIa).
     - Surgical treatment is completed via peritoneal wash and lymph node sampling for microscopic assessment of the extent of the disease.

B. <u>Unilateral salpingo-oophorectomy:</u>

May be indicated in patients with stage Ia only when the patient is young and preservation of fertility is desired.

2. *Advanced stage ovarian cancer:*

A. <u>Initial debulking(primary cyto-destructive surgery):</u>

- Includes TAH-BSO + omentectomy + excision of pelvic masses and peritoneal deposits > 1-2 cm + segmental bowel resection if involved within the tumour mass.
- Aims to improve all 1ry cancer and all metastatic disease within the pelvic and peritoneal cavities.
- Advantages:
  - Improves survival in patients with advanced disease.
  - Improves response to chemotherapy.

B. <u>Interval debulking:</u>

Chemotherapy prior to debulking to minimize tumour bulk and control ascites to allow for subsequent more complete radical surgery.

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3. Mention key points in ovarian cancer.

- Epithelial ovarian cancer is **the Commonest type**
- Diagnosis is based on:
  a. History.
  b. Clinical examination.
  c. U/S findings.
  d. Tumour marker assessment.
- Other investigations as: abdominal U/S, Chest X-ray, C.T. scan, MRI, IVP, barium follow through, upper and lower endoscopy are helpful in defining the extent of the disease prior surgery.
- Important criteria of malignancy includes:
  a. Solid or mixed solid and cystic consistency.
  b. Bilaterality.
  c. Presence of ascites or extra-cystic papillae.
  d. Fixation.
- Tumour markers include:
  a. CA125 in epithelial cancers.
  b. HCG in choriocarcinoma.
  c. LDH in dysgerminoma.
  d. AFP in EST.

  They are helpful in diagnosis and follow up after surgery.
- Tumours Stage I carry the best prognosis, however unfortunately most ovarian cancer will be diagnosed at stage II or III due to absent specific symptoms in early disease leading to poor prognosis.
- A staging laparotomy is indicated in every malignant ovarian tumour regardless it`s staging.
- During laparotomy removal of main ovarian bulk is done together with peritoneal fluid cytology, omentectomy, and lymph node sampling in order to assess the extent of disease, the prognosis, and need of adjuvant TTT.

- Standard surgical approach entails performing a TAH-BSO and infra-colic omentectomy.
- Primary cytoreductive surgery in advanced disease aims at removal of maximum tumour bulk leaving tumours residues less than 2 cm to facilitate post-operative chemotherapy.
- The majority of malignant ovarian tumours well require adjuvant post-operative chemotherapy. Some cases benefit from radiotherapy.
- Malignant germ cell tumours can be managed conservatively by unilateral salpingo-oopherectomy in cases with stage IA to preserve fertility as most unilateral tumours occurs in young patients.

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8. MISCELLANEOUS

Scheme for last part of book (imaging - endoscopy - operations - instruments):
"Indications- Complications-Contraindications-Timing- General scheme"

i. ***Indications*** *are always diagnostic and therapeutic:*

Try to remember the topics of our book by order and ألف منها

- Embryology
- Amenorrhea
- Bleeding
- Infertility
- Neoplasm
- Contraception
- Infections

Example:

Hysteroscopy	
Diagnostic	**Embryology:** Septate
	Amenorrhea: Asherman
	Bleeding: AUB
	Infertility** Most imp.
	Neoplasm SMM
	Contraception --
	Infection --
	Others: Recurrent abortion
Therapeutic	**Embryology:** Resection of septum
	Amenorrhea: Removal of adhesions in asherman
	Bleeding: Polypectomy, endometrial ablation
	Infertility: Tubal catheterization for corneal obstruction تسليك الأنابيب من داخل الرحم
	Neoplasm: SM Myomectomy
	Contraception: Removal of missed IUCD, sterilization (sclerosing agent in Cx or silicon plugs in tubes)
	Infection --
	Others --

ii. ***Contraindications*** *are always undiagnosed vaginal bleeding (fear of malignancy), amenorrhea (fear of pregnancy), PID (fear of spread of infection).*

And something special in each procedure:

e.g. Laparoscopy:

- Morbidly obese, cardiac or chest condition (Trendelenburg تقتله)
- Organomegaly, large uterus (more than 16 weeks) عشان احتمال اخرم
- Adhesion (for example due to repeat CS) (high incidence of injuries)

iii. ***Complications*** *are always early and late:*

Early:

- Hge, Inf, Injury and anasthesia
- And *injury* maybe أحمر، أصفر، بني. Blood vessel, ureteric or intestinal.
- *Injury* maybe due to مشرط أو مكواة. Direct or diathermy.

Late:

DVT and PE if long surgery (hysterectomy) or Rupture uterus if perforation..

And something special in each procedure:

- **Hystroscopy:** fluid overload and electrolyte imbalance
- **Laparoscopy**: Air embolism and surgical emphysema
- **Hystrectomy:** wound complications as infection, keloid and hernia
- **HSG:** anaphylaxis and oil embolism

iv. ***Timing:*** *Gyne: postmenstrual. Obs: 3-6 months postpartum.*

With few exceptions:

Gyne: Premenstrual:

- Biopsy in detection of ovulation and TB diagnosis.
- Endometriosis in laparoscopy (lesions are at largest size)
- Isthmography in HSG. (Maximum effect of PRG on cx)

Obs:

- Ovarian swellings are removed intraoperative in CS or 1 week after vaginal delivery.
- Ectopic وقت ما يظهر مالوش معاد محدد

v. ***General scheme for any operation:***

1. **Position:**

Lithotomy or dorsal

2. **Anasthesia:**

General or regional

3. **Bimanual examination:**

To know size and position of uterus

4. **Sterilization with betadine**
5. **Catheterization with foley's catheter**
6. **Auvard's self retaining speculum to open vagina**
7. **Vulsellum to grasp cx**
8. **Sounding to know uterine length**

ثم نبدأ العملية

Diagnostic and operative procedures

1. **Possible diagnostic applications of US in gynaecology.**
    1. Evaluation of genital anomalies
    2. Evaluation of pelvic masses
    3. Evaluation of lower abdominal pain & inflammatory processes especially in patients with muscular guarding or rigidity.
    4. Monitoring of follicular growth as part of infertility workup.
    5. Localization of I.U.D. (either post insertion or in occasions of missed device)
    6. Diagnosis of abnormal uterine bleeding
    7. Diagnosis and evaluation of uterine fibroids (number, size, site, type, relation to the cavity)
    8. Measuring endometrial thickness.
    9. Diagnosis of ovarian cysts and neoplasms.
    10. Diagnosis of ectopic pregnancy.
    11. Evaluation of urinary incontinence and residual urine volume.
    12. Sonohysterography for tubal patency.
    13. Can differentiate between benign and malignant conditions with a reasonable degree of efficacy.

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2. **Discuss value of transvaginal ultrasound in assessment of AUB during child bearing period (September 2016).**

    It is the gold standard investigation to diagnose different causes of AUB as:
    1. Diagnosis and evaluation of uterine fibroids (number,size,site,type,relation to the cavity).
    2. Measuring of endometrial thickness.
    3. Diagnosis of ovarian cysts and neoplasms.

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3. **Indications of laparoscopy (June 2005, 2013, September 2012)**

A. Diagnostic laparoscopy:

1. Infertility whether primary or secondary .
2. Chronic pelvic pain.
3. Diagnosis of pelvic endometriosis.
4. Diagnosis of some undiagnosed pelvic masses .
5. Assessment of congenital anomalies of u & tubes.
6. Follow up after radical surgery for malignancy .

B. Operative laparoscopy:

a. Tubal surgery:

- Management of tubal obstruction and adhesion by adhesolysis, salpingostomy, fimbrial dilatation and salpingectomy
- Management of ectopic pregnancy.
- Removal of hydrosalpinx or pyosalpinx.
- Tubal sterilization.

b. Ovarian surgery:

- Selective removal of ovarian masses with no or minimal risk for malignancy by cystectomy, ovariotomy or oophorectomy.
- Drilling of polycystic ovarian disease in cases resistant to medical treatment.

c. Uterine surgery:

- Myomectomy for myomas of small number and moderate size <9 cm.
- Hysterectomy whether total or subtotal is feasible in selective cases.

d. Endometriosis:

Variable procedures can be performed including laser ablation, fulguration and diathermy cauterization of endometriotic foci, pelvic adhesiolysis, endometrioma excision and adnexal removal and hysterectomy

d. Pelvic floor relaxation.

e. Some selective radical procedures for malignancy.

f. Omental and intestinal adhesiolysis to relieve variable degrees of pain resulting from adhesions of previous surgery.

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4. Value of laparoscopy over HSG (June 2006).

Advantage of operative laparoscopy:

1. Minimal hospital stay and early return to work.
2. Minimal patient discomfort.
3. Minimal patient adhesion and therefore minimal iatrogenic infertility.
4. Better cosmetic results.
5. Rare wound complications.
6. Allow proper inspection and excludes the need for what is called exploratory laparotomy.

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5. Complications of H.S.G.

1. Shock.
2. Oil embolism.
3. Disturbance of an undiagnosed pregnancy .
4. Flaring up or introduction of infection.
5. lodine allergic reactions.
6. Intravasation (the oil passes into the uterine vessels during injection which is mainly lymphatic & rarely venous).

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6. Indications of hysteroscopy (September 2009).

A. Diagnostic :

1. Infertility; Variable findings may be occasionally found such as polyps, synechiae, submucous myoma, congenital anomalies e.g. septate uterus.
2. Habitual and recurrent abortions.
3. Irregular uterine bleeding

B. Operative:

1. Polypectomy.
2. Resection of uterine septae.
3. Resection of a submucous myoma.

4. Division of intrauterine synchiae.
5. Removal of a missed IUD.
6. Resection or coagulation of the endometrium.
7. Intrauterine tubal catheterization for corneal obstruction.
8. Sterilization by injecting a sclerosing gent or a plug to the cervix.

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7. Causes of symmetrically enlarged uterus (September 2009).

1. Pregnancy
2. Metropathia haemorrhagica
3. Single submucous or single interstitital fundal fibroid
4. Diffuse adenomyosis
5. Subinvolution of the uterus (in the puerperium)
6. Malignant tumours: as adenocarcinoma, sarcoma or choriocarcinoma
7. Pyometra and haematometra:
   - In pyometra, there is lower abdominal pain and fever. The uterus is, tender and cystic.
   - In haematometra there is amenorrhea, recurrent lower abdominal pain every month, atresia at or below the cervix.

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8. Causes of cervical enlargement (September 2011)

1. Chronic cervicitis.
2. Congestion of the cervix (e.g. in pregnancy and prolapse).
3. Congenital elongation of the portio vaginalis.
4. Elongation of the supravaginal cervix in cases of vagino-uterine prolapse.
5. Cervical tumours whether benign (e.g. fibroid) or malignant (e.g. carcinoma).
6. Distension of the cervix by an extruded uterine polyp.
7. Some specific inflammations e.g. tuberculosis, bilharziasis and syphilis.
8. Cervical abortion.
9. Cervical ectopic pregnancy.
10. Endometriosis of the cervix.

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9. Gynaecological causes of acute abdominal pain (September 2011)

1. Disturbed ectopic pregnancy.
2. Acute salpingitis.
3. Torsion, rupture or haemorrhage in an ovarian cyst.
4. Red degeneration in a fibroid or torsion of a pedunculated subserous fibroid.
5. Haemorrhage in a corpus luteum or rupture of a corpus luteum cyst.

   The above mentioned gynaecological causes should be differentiated from the non gynaecological causes of acute lower abdominal pain e.g. acute appendicitis, acute pyelitis, diverticulitis, etc. Acute upper abdominal pain may be due to cholecystitis, pancreatitis etc ...

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10. Uses of uterine sound (September 2011)

1. Degree of supravaginal elongation of the cervix in cases of prolapse.
2. Diagnosis uterine hypoplasia (the uterine length is subnormal and there is alteration in the of ratio between length of the cervix and body).
3. Measurement of the length of the uterine cavity preliminary to dilatation.
4. Measurement of the uterine length before introduction of I.U.D.
5. Diagnosis of the direction of the uterus.
6. Diagnosis of cervical stenosis (inability to introduce the sound).
7. Abnormality inside the uterus as polyp or septum.
8. Differentiation between chronic inversion and fibroid polyp bulging through the cervix. In the latter case, the uterine length is normal.
9. Detection of retained I. U. C.D.
10. Diagnosis of endocervical carcinoma.

Clinical gynaecology and history

1. **Indications of rectal examination in gynaecology (sep 2009, june 2010, 2011)**

1. Examination of **virgins.**
2. In **cancer cervix** , to detect extension to parametrium along the uterosacral ligament.
3. In cases of **prolapse** to detect presence of rectocele and exclude presence of enterocele.
4. Cases with **complete perineal tears** if diagnosis is not sure by inspection alone.
5. Cases with **mass in Douglas pouch.**
6. Cases with **postmenopausal bleeding** (piles, cancer rectum) .
7. Any patient complaining of **rectal symptoms.**

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2. **Points to be determined during bimanual examination (2012)**

Mnemonic:

vagina, muscles, pelvic bones and cervix باحس for PV alone
و لما أوصل لل cervix أحسها و توجهاتها و أحركها و أشوف الlevel
size, tenderness, consistency أحسها يعني أشوف
position of external os .. توجهاتها يعني mobility أحركها يعني
لو uterus هيبقى نفس الكلام بس زود اخواتها الadnexa و وانت بتطلع إيدك متنساش تشوف الdischarge

1. *Uterine size, shape, consistency, and contour:*

   The uterus is a pear shaped organ with firm consistency, and smooth outer contour. It normally measures nearly 7.5 x 3.5 x 5.5 cm in Long/AP/Transverse, diameters. It may be felt with difficulty in women with marked trunk obesity.

2. *Uterine Position:*

   The uterus is commonly Anteverted flexed (AVF) in position, with the external os pointing towards the posterior fornix. In nearly 20% of cases the uterus is retroverted flexed (RVF), with the external os pointing towards the anterior fornix.

3. *Uterine tenderness and mobility:*

   The uterus is slightly tender when squeezed between the two hands. It is normally mobile, but might be fixed with adhesions or pelvic masses.

4. *Presence of Adnexal Tenderness, Fullness, or Masses:*

   - The normal tubes are never palpable even in the very thin patient, while the normal ovaries can be felt in the thin patient, in absence of pelvic tenderness .
   - Presence of adnexal tenderness, fullness, or masses (cystic or solid), is always abnormal, and should raise suspicion for presence of pelvic pathology as; PID, ectopic pregnancy, and tubo-ovarian masses.

5. *Evaluation of vaginal discharge:*

   The amount and character of vaginal discharge, if present, are noted on the examining fingers while being withdrawn outside the vagina at the end of vaginal examination.

Final exam – June 2018

1. **A 28-year old woman with 3 living children wants to have contraception. She refuses the intra-uterine contraceptive (IUD) and prefers a hormonal method of contraception:**
   - Enlist the available hormonal method that can be given to this woman.
   - Enumerate the side effects of hormonal contraception.
2. **An infertile couple is investigated for 2 years infertility. The woman has normal mid-luteal serum progesterone and hysterosalpingogram. The husband is waiting for the result of his semen analysis:**
   - What are the normal values of different parameters in the semen analysis?
   - Define the different terms of abnormal semen parameters
3. **A 27 years old woman was diagnosed to have endometrioma and pelvic endometriosis. After surgical removal of the endometrioma she is put on medical therapy for her remaining endometriotic foci:**

   Enumerate different pharmacological treatment options available to be given to the woman with duration and possible side effects of each option.
4. **During a routine pap smear, CIN Ⅱ was diagnosed.**
   - How to confirm the diagnosis?
   - What are the different treatment modalities?
5. **In evidence bases medicine (EBM)**
   - List the types of clinical studies.
   - Give the criteria of randomized controlled trials.
6. **In assisted Reproductive Technology cycles:**
   - List the steps that are followed in an IVF cycle.
   - List the factors that affect the success rate of IVF / ICSI cycles.
   - List the complications of IVF / ICSI cycles.
7. **In the outpatient clinic your bedside manners should respects the code of ethical conduct:**
   - Mention the main points of this code.
8. **The vagina has natural defense mechanisms against genital tract infections.**
   - List these factors.
   - Explain the factors that predispose to vaginal infections.
   - List the common types of vaginitis in the childbearing period
9. **List the non-neoplastic ovarian swellings.**
   - Explain the means of management of these swellings.
10. **Define menopause and describe its clinical features.**

Final exam – August 2018

1. **A 39-year-old woman, G5, P4, all her deliveries were vaginal deliveries. She came to your clinic for advice on contraception. She refuses hormonal method of contraception.**
   A. **What is the most effective method of contraception that you advise her to use?**
   B. **What are the side effects and complications of your choice of method of contraception?**
2. **Anovulation is one of the most common causes of female infertility.**
   Describe the different methods of detection of ovulation.
3. **A mother noticed that the breast of her 5-years child girl started to enlarge, with some pubic hair appearing at the mons pubis.**
   A. **Describe the different types of precocious puberty.**
   B. **Describe how to diagnose each type.**
4. **A 39-year-old female, G4, P4, all by vaginal delivery. She has cysto-rectocele diagnosed 3 years ago. Few weeks ago, she noticed that she could touch her cervix easily in the vagina.**
   A. **Classify the stages of uterine prolapse.**
   B. **Describe the lines of treatment of this woman.**
5. **A 26-year-old nulliparous woman discovered that she has fibroids of the uterus upon doing an abdominal ultrasound because of vague abdominal pain.**
   A. **Enumerate the possible gynecological symptoms that may be present with fibroid uterus.**
   B. **Describe how fibroid uterus may affect her chances to get pregnant and deliver a live full-term baby.**
6. **A 65-year-old postmenopausal woman complained of abnormal vaginal bleeding. She performed fractional curettage that diagnosed endometrial carcinoma.**
   A. **What are the further investigations that you would request for this patient?**
   B. **What is the best surgical treatment in this case? Enumerate the surgical staging of this type of malignancy.**
7. **A young recently-married woman presenting with acute bilateral lower abdominal pain is suspected to have pelvic inflammatory disease (PID).**
   A. **Describe the CDC criteria of diagnosis.**
   B. **Enumerate the lines of treatment of PID.**
8. **A 26-year-old woman came complaining of primary infertility since her marriage 3 years ago due to bilateral hydrosalpinx. You are preparing for an IVF cycle.**
   A. **What is the special preparatory step in this particular patient before proceeding to IVF.**
   B. **List the possible complications in her IVF cycle.**
   C. **If there is associated polycystic ovarian disease, what is the most serious potential risk in induction? Describe how can you prevent it.**
9. **The pituitary gland is the maestro of the endocrine glands of the body.**
   A. **Describe the anatomy of the pituitary gland.**
   B. **List the hormones secreted by the pituitary gland.**

10. **Ultrasound is considered the most commonly used diagnostic tool in gynecology. Enumerate the diagnostic applications of ultrasound in an infertile woman.**